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THE
LARYNGOSCOPE.

VOL. LXIII JULY, 1953 No. 7

THE IDENTIFICATION AND CLINICAL SIGNIFICANCE OF LARGE PHAGOCYTES IN THE EXUDATES OF ACUTE OTITIS MEDIA AND MASTOIDITIS.*†‡§

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A study of the cytology of middle ear and mastoid suppurations has revealed the occurrence in certain cases of large phagocytic cells; in fact they are so large that they may be well termed "giant phagocytes". To our knowledge they have not been described previously in aural exudates. This report is concerned with the identification and description of these cells with their coexistent cytology and an analysis of their clinical occurrence and resulting diagnostic significance.

The cytology of ear secretions has been studied in general, and from special points of view, by relatively few investigators. References, without any detailed descriptions, to the usual cells found in pus, namely neutrophilic leucocytes, lymphocytes, monocytes and eosinophiles, are found in the German literature of fifty years ago (Politzer, Bruhl).[14] In 1926 Ts'en, Shih-Ping[22] reported seven aural smears with case histories and suggested that the differences in cytology

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†Read at the 57th Annual Meeting of the American Laryngological, Rhinological and Otological Society, New Orleans, La., April 29, 1953.

‡Grateful acknowledgment is made to my wife, Marian P. Bryan, who found the aural phagocytes described here and who did the laboratory work and drawings incorporated in this paper.

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were due to the duration of the discharge. He noted that the neutrophiles were more degenerate in the chronic cases than in the acute ones. Fewer bacteria were observed in the acute conditions. Hesse,[8] in the following year, 1927, reported a study of fresh exudates from middle ear suppurations in which he attempted to correlate the phagocytic power of supravitally stained leucocytes with the severity of the infection. He used the Snyderhelmishe dye, a preparation of trypan blue and congo red. He found that the cells died rapidly with this stain. In our experience the dye was very toxic for the cells and did not give sufficient time to observe them in the living condition.

L. W. Dean, Sr.,[4] in summarizing the laboratory aids that were helpful in otolaryngologic diagnosis emphasized the importance of the aural smear. He mentioned that the state of degeneration of the neutrophiles in ear discharge in acute otitis media suggested the severity of infection. The greater the degeneration, the more active was the infection.

Dean, Jr., and Pfingsten,[3] in 1933, studied the cholesterol content of aural exudates and described the cytology of twenty-six cases of chronic otitis media as well as nine cases of acute otitis media. Cholesterol crystals, degenerated neutrophiles, bacteria, fatty degenerated epithelial cells and mucus were found in the chronic cases. One of these showed an increase in eosinophiles. In the acute cases the fatty degenerated epithelial cells were less numerous and the neutrophiles were increased over those in the chronic cases.

In regard to allergy of the ear, Hansel[7] suggests that the finding of eosinophiles in the aural discharge indicates chronicity of infection. Proetz[15] studied the secretion in a case of allergic middle ear disease and did not find eosinophiles at the time of paracentesis. Dohlman[5] worked on the problem of eosinophiles in the discharge of chronic otitis media and a few years later in 1947 Koch,[10] working in his department, made a very complete and detailed study concerning the finding of eosinophiles in the secretions from chronic otitis media. He concluded that in the cases exhibit-

ing an increase of eosinophiles, allergy could be demonstrated, and that the healing time was more protracted. The secretions in these cases were clear to serum-like in appearance and very viscous in character.

Senturia, Matthews and Adler[21] showed the importance of aural cytological studies in differentiating the many types of external otitis.

House[9] used the Papanicolaou stain in studying the cytology of chronic aural discharge for the diagnosis of malignancy.

Phagocytes or histiocytes have been found and studied in various aural tissues. Russi[16] vitally stained guinea pigs with 1 per cent trypan blue and found a few histiocytes in the decalcified sections of the tympanic membrane, most of which were on the outer surface and toward the periphery. He mentioned that they were sparsely scattered about the round and oval windows. In the Eustachian tube a few histiocytes occurred in the submucosa; there were more in the perichondrium, and they increased in number toward the tympanic end of the tube. Fenton and Larsell[6] working with this problem in 1932, made their observations on cats vitally stained with 1 per cent trypan blue. Histiocytes were found sparsely scattered along the Eustachian tube and there were still fewer in the tympanic mucosa, mainly near the capillaries. In one human case, killed by accident, they found histiocytes from the mouth of the Eustachian tube into the tympanum, the numbers increasing with the distance from the throat. Schwarz[23] examined 202 fetal and newborn temporal bones, in 58 of which there was a slight otitis media. In the third and fourth month the histiocytes comprise 10 per cent of the connective tissue cells, then they rapidly increase and appear in the tympanic cavity. Liveriers[11] made an anatomic and histologic study of the reticulo-endothelial system of the inner ear in guinea pigs. The histiocytes were found in the spiral ligament and in the periosteum of the tympanic section.

The literature on histiocytes in general is voluminous and well summarized in textbooks on cytology.[2,13] It is applicable here only indirectly to the extent that it helped in the original identification of the cells.

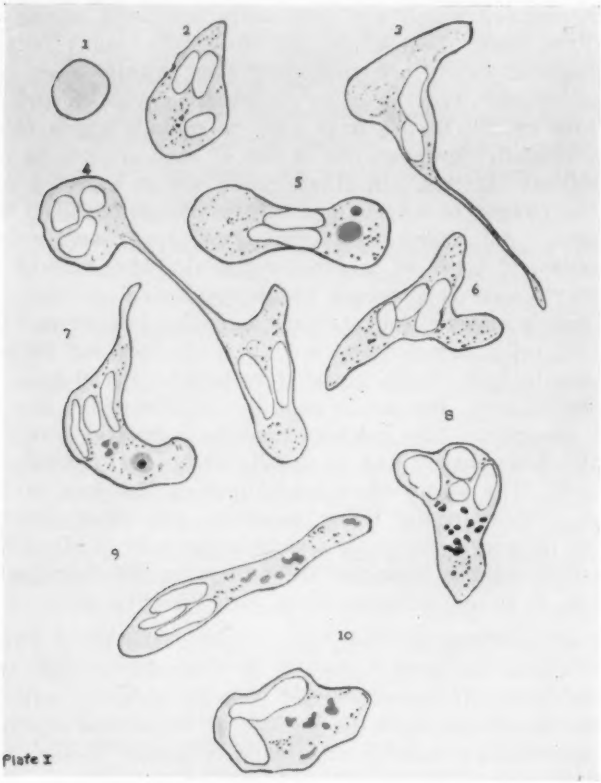
Summarizing the above references on aural phagocytes, it is found that histiocytes are present in the soft tissues of the ear in anatomical studies. Attempts have been made to evaluate the severity of clinical otitis media by studying the viability of leucocytes in aural exudates, but these observations have not included the identification of the phagocytes or their diagnostic significance in the discharge.

The giant phagocytes forming the subject of this report were first seen by us when the method of supravital staining, as developed by Sabin[17] for the study of living human blood cells, was applied to aural secretions. This technique was used to study 90 of the 227 cases of acute otitis media and surgical mastoiditis included in this report. It was thus possible to observe various conditions of cellular activity such as phagocytosis, motility and contractions of the cells. The method enables one to use certain non-toxic dyes for staining, such as neutral red and Janus green in weak dilutions without killing the cells. A chemically clean slide is flooded with the dye and allowed to dry. When the cells in a drop of exudate come in contact with the dye film there is just enough dye to stain the cellular cytoplasm, not the nucleus, without endangering the life of the cells within a period of two to four hours. Nuclear staining is evidence of cell death. The films were sealed with paraffin and studied in a warm stage at body temperature. The exudate should be as fresh as possible since old pus contains only dead and disintegrated cells. It was found satisfactory to obtain the secretions with an appropriate suction tip such as a small sinus cannula with bulb. After cleansing the external canal, gentle suction usually yielded viable material. Dry smears stained with Wright's were made from the same material for comparison and in order to have a permanent record of the cytology.

In the living condition with supravital staining, the giant phagocytes are striking. They vary in size from 20 to 50 microns in diameter and the cytoplasm may be filled or partially filled with granules and vacuoles of varying size and colors of neutral red. They may also contain some clear vacuoles which are irregularly scattered throughout the cytoplasm. Leucocytes, red blood cells, many bacteria and cellular debris are frequently seen within these cells. In exceptional fields as many as five neutrophiles and two to three red blood cells have been noted in a single phagocyte. Usually, however, one or two of each are visible with or without bacteria. In others there are no ingested cells, but the phagocyte may be partially or completely filled with bacteria. All degrees of stimulation are noted by the amounts and types of ingested material. The body of the phagocytic cell is generally roughly spherical or oval, but at times it may be elongated and irregular in contour. The limiting cellular membrane may be indistinct and often it appears to bulge with ingested material. Pseudopods are frequently seen, but active motility of these cells has not been observed. The nucleus may be spherical, ovoid or slightly bean-shaped and is usually eccentrically located in the cell. The larger phagocytes may exhibit two to four nuclei, which, in the living condition, are often obscured by the phagocytosed material within the cell. Plate 3 Nos. 18 and 19 of the drawings show them in the living state. Figs. 8, 9, 10 and 11 show them with Wright's stain.

At other times, in fresh preparations, the giant phagocytes appear as large spherical or irregular masses completely filled with smooth round globules of lipoid material. In this functional state the nucleus or nuclei are obscured, and the limiting cellular membrane is barely visible. If a small amount of Sudan III which is an indicator of lipids is added, the globules filling the cytoplasm stain orange. They have been called "lipoid phagocytes". Plate 2 of the drawings shows them in the fresh condition with a red blood cell for comparative size. Figs. 2 and 6 show them in Wright's stain. Under polarized light the globules exhibit double refraction with one or two liquid crystals of choles-

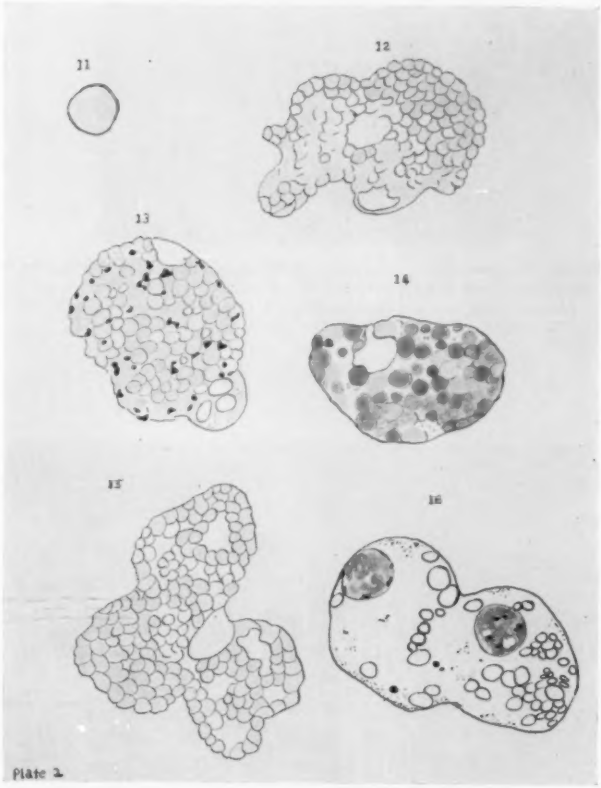
terol in the form of a Maltese cross, previously described in aural smears by Dean, Jr. and Pfingsten.[3] It is thus possible at times to demonstrate the presence of cholesterol within these cells.



Neutrophiles from acute otitis media (supravital films) showing amoeboid movement: 2, 3, 4, 5, 6 are stained with neutral red; 7, 9 and 10 have phagocytosed carmine; 8 contains carbon particles. 1. shows a red cell for size.

The fact that the histiocytes take part in the storing of fats and lipids in the tissues was shown by Anitschkow,[1] Zinserling[23] and Schönheimer.[19] After feeding animals with

cholesterol the histiocytes all over the body are reported to be filled with large quantities of droplets of anisotropic cholesterol esters. Maximow[13] states that fat and lipoid



A group of lipoid histiocytes: 12 and 15 are unstained living cells; 13 is supravitally stained with neutral red; 14 is stained with Sudan III; 16 is stained with Wright's.

inclusions are of common occurrence in these cells linking them very closely with fat metabolism. He also mentions that histiocytes in their various locations show more or

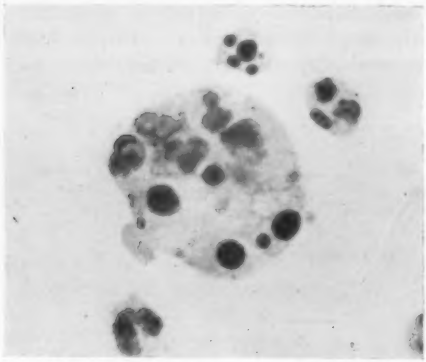


Fig. 1*. Typical giant phagocyte with ingested neutrophiles and cellular fragments from discharge of acute purulent otitis media.

*All photographs were taken under oil immersion with a 95X objective and a 15X ocular. Wright's stain was used.

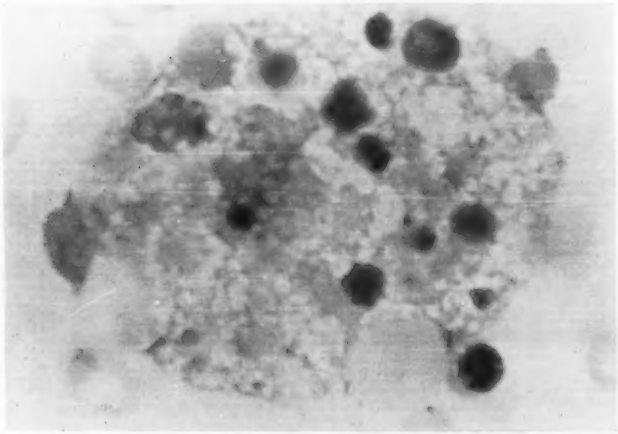


Fig. 2. An especially large giant histiocyte from a case of acute coalescent mastoiditis. Cell contains numerous bits of cellular debris, red blood cells, very many vacuoles and nucleus somewhat obscured by the phagocytosed material. Note that limiting cellular membrane is bulging and is slightly indistinct.

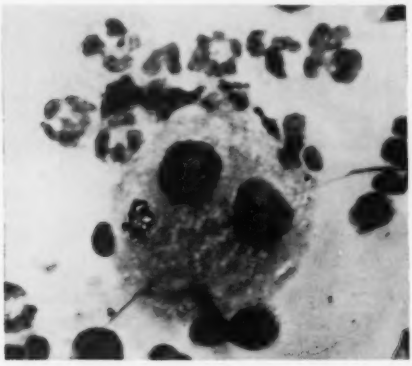


Fig. 3. Double nucleated large phagocyte in a group of neutrophiles and lymphocytes. Specimen from mastoid antrum in a case of acute coalescent mastoiditis.

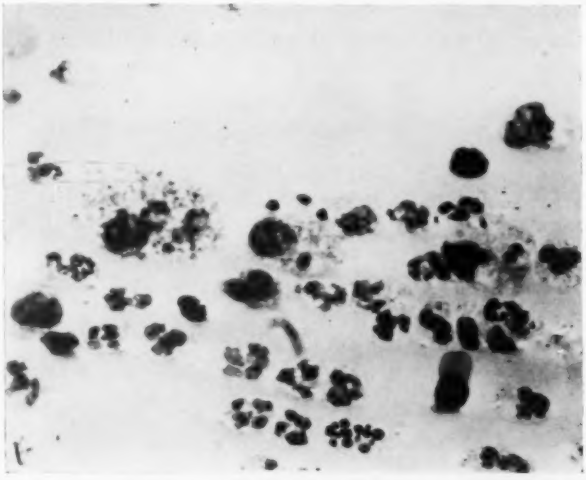


Fig. 4. Group of three moderately large phagocytes from aural discharge of acute mastoiditis. Note eccentric rough nuclei, ingested bacteria and other debris.

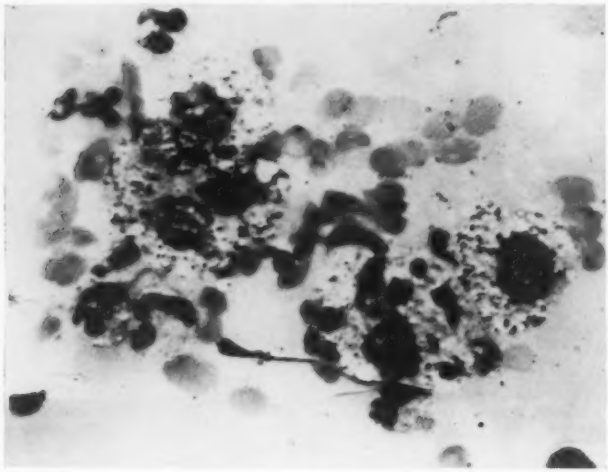


Fig. 5. Four giant phagocytes from mastoid cavity of acute coalescent mastoiditis showing large numbers of ingested bacteria. Huge cell to lower right has double nucleus. Adhering to this cell is a smaller one exhibiting rough irregular nucleus, vacuoles and indistinct cellular membrane. Two histiocytes to upper left show similar clumps of engulfed material.

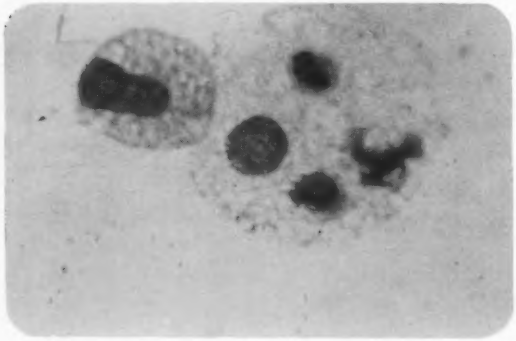


Fig. 6. Two lipoid phagocytes from discharge of radical cavity with continuous acute infection since original otitis about two years before. Note foamy cytoplasm where lipoid globules have been dissolved by staining and ingested material in the larger cell.

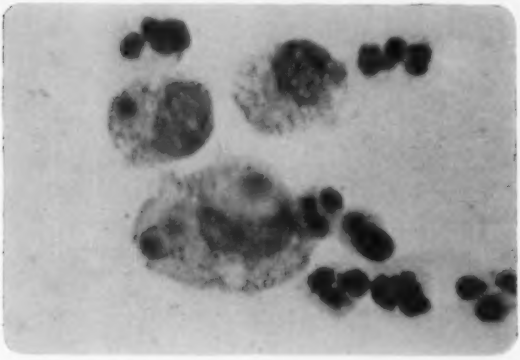


Fig. 7. These cells are from same radical cavity described in Fig. 6. Two cells are smaller monocytic phagocytes. The comparison in size with neutrophiles is interesting.

less typical structural differences and that their microscopic appearance is strongly influenced by their functional conditions; this certainly is verified in our material.

The largest giant phagocytes, when present in these aural smears, constitute the most unique element of the cytological

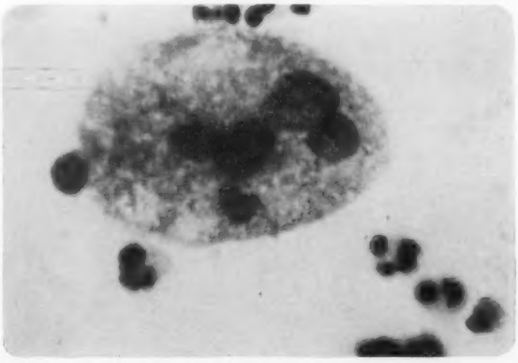


Fig. 8. Quadruple nucleated ovoid giant phagocyte with foamy cytoplasm and distinct cellular membrane from aural discharge of an acute exacerbation of chronic otitis media and mastoiditis. Size may be compared with neutrophiles in the same field.

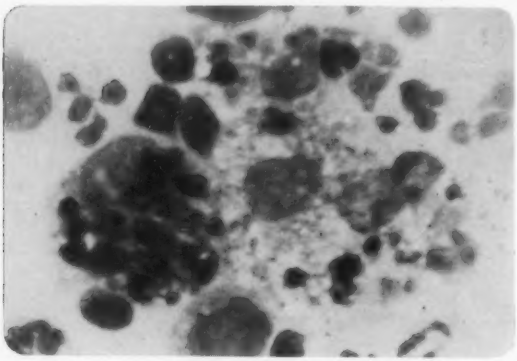


Fig. 9. Two giant histiocytes from the same aural discharge as Fig. 8. Note coarse heavy ingested material and particulate matter colored dark purple in contrast to the lighter nuclei. The larger cell exhibits a foamy cytoplasm.

pictures. Actually in all the smears in which they are found, there are numerous smaller ones of all gradations in sizes and with various amounts of ingested material (see Fig. 7). The smallest are but slightly larger than the true blood monocyte. These phagocytes measure 15 to 25 microns in diameter. They are easily distinguished from the true monocyte which

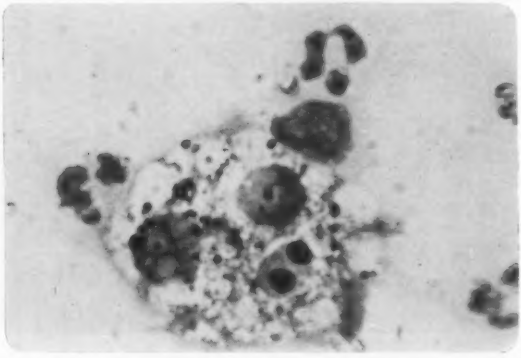


Fig. 10. Double nucleated giant histiocyte with vacuolated foamy cytoplasm containing bacteria. One ingested neutrophile is also visible. Material is from an acute exacerbation of a chronic otitis media.

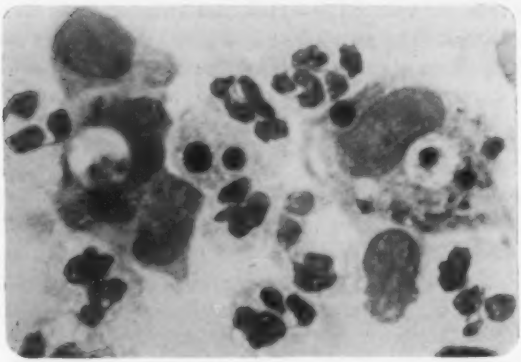


Fig. 11. From same case as Fig. 11. Note bean-shaped nucleus of phagocyte and monocyte and neutrophiles in the field.

measures 12 to 15 microns, by their smaller ovoid or spherical nucleus, usually eccentric, and by their irregular vacuoles and variety of ingested material in contrast to the finely granular cytoplasm of the monocyte. These "transitional" phagocytes correspond to the "clasmatocyte" in the connective tissues of Sabin's classification.[18] When the secretions

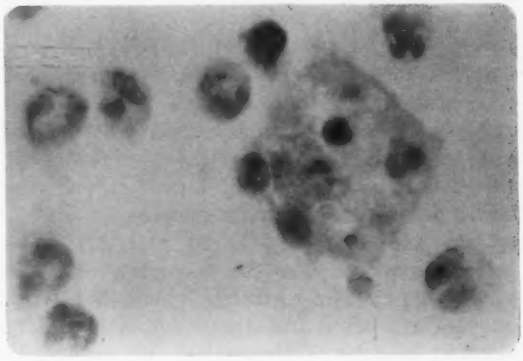


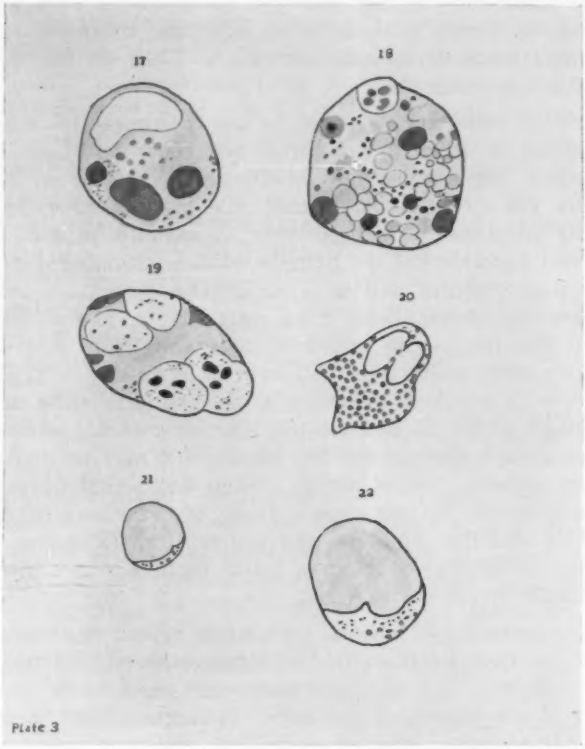
Fig. 12. Single giant phagocyte with ingested material and red blood cells from mastoid antrum. The acute infection was complicated with a temporal abscess on the same side. Note neutrophiles in the field.

contain the largest phagocytes the smaller ones are always present in varying numbers; however, the smaller ones have been present in smears without the giant ones. All of these cells are obviously phagocytes and are found in pus from mastoiditis.

In the supravital preparations the neutrophilic leucocytes are the most numerous element. They exhibit active motility with protoplasmic streaming. Their cytoplasm stains lightly with neutral red (see Plate 1, Nos. 2, 3, 4, 5 and 6). In acute otitis media, during the first few days of the infection, about 40 to 65 percent of the neutrophiles react to neutral red in the cytoplasm, whereas the dead ones exhibit nuclear staining and are non-motile. It has been demonstrated that the same vacuoles of a polymorpho-nuclear neutrophile which react to neutral red will phagocytose particulate matter such as carbon particles of India ink and carmine (see Plate 1, No. 7). This may be done by adding carbon particles to the fresh preparation and counterstaining with neutral red. A black particle may then be seen with a vacuole stained with neutral red. Plate 1, Nos. 8, 9, 10 show phagocytosis of carmine and carbon particles. Such an experiment tends to prove that supravital neutral red staining is a fairly accurate indication for phagocytic activity. The neutral red dye was much more satisfactory than the Snyderhelmische dye that was used by Hesse.[8]

Lymphocytes both large and small are observed in these films. They vary in size and nuclear detail from the mature blood lymphocytes to the immature blast forms which are often seen in mastoiditis. They show a few neutral red granules in the cytoplasm and if Janus green is used the mitachondria are seen as minute granules or short rods in the cytoplasm (see Plate 3, Nos. 21 and 22). They may exhibit slight motility but do not show any noticeable phagocytic activity. The irritated stimulated lymphocytes and monocytes are often indistinguishable in inflammation. The distinction may be of theoretical importance, but from a clinical viewpoint it is unimportant except for the fact that large numbers of such irritated younger forms indicate

severity of infection. This is especially true when they accompany the giant phagocytes. Lymphocytes alone, without the other inflammatory cells, are frequently found in chronic mastoiditis.



Cells of acute middle ear exudates (supravital films): 17 is a monocyte with large vacuoles of neutral red; 18 shows a phagocyte with neutral red and lipoid vacuoles; 19 is a phagocyte with included neutrophiles; 20 is an eosinophile with neutral red stain; 21 and 22 are lymphocytes with neutral red and Janus green.

Eosinophiles, in most of the acute aural secretions are sparsely scattered. An increase was noted in five cases.

They may exhibit active amoeboid motility, but they have never been seen to absorb particulate matter in the fresh preparations (see Plate 3, No. 20).

Another interesting cell seen in acute aural infections is the large basophile. This cell is never noted in more than small numbers. The granules are heavy coarse dark purple or red in Wright's stain, often covering the nucleus and spilling outside of the cell membrane. Their significance at this time is unknown.

It is not necessary, however, to use the supravital staining technique in order to see all of the cells that have been described. With rapid light staining in Wright's, when the smears are air-dried, not flamed, the giant phagocytes are readily identified, although their appearance is somewhat different from that of the living condition. Staining dissolves the lipoid globules and as a result the cytoplasm usually appears vacuolated, thus, these cells have a foamy appearance. The phagocytosed debris, cells and bacteria are accurately seen within the histiocyte in Wright's stain. It is possible to overlook them in a slide if the slide is not examined under oil because the limiting cellular membrane is not always distinct and the phagocytes may be mistaken for an accumulation of debris. When one actually observes the phagocytes in these slides there is never any doubt as to their identity. Since we can use routine stains, we have a simple laboratory method to detect them which is directly applicable to clinical material.

The clinical significance of these giant phagocytes was demonstrated first by the study of smears taken from mastoid cells. Every case of acute coalescent mastoiditis showed many, if not masses, of such cells. In contrast, they were not found in quiescent chronic mastoiditis. Since it was demonstrated in both the fresh and dry stained preparations that they were associated with osteitis, it seemed likely that they would have some diagnostic value when found in aural discharge.

Smears from various types of discharge from the ear were then collected from both clinic and private patients. Over

2000 smears were studied. Of the 277 cases examined, 227 were of acute otitis media or mastoiditis, 33 were chronic mastoiditis, 13 were infected radical cavities and four were mastoid abscesses. They have been collected since 1933 and include all the cases in which smears were obtained. There are three main periods represented: the first, a pre-antibiotic one from 1933 to 1934; the second from 1939 to 1940 when sulfonamides were first introduced and were used timidly; the third from 1947 to the present when antibiotics were used freely. In Table 1 the number of giant phagocytes found in the cases of acute otitis and mastoiditis is plotted against recovery.

TABLE 1. THE PRESENCE OF GIANT PHAGOCYTES IS PLOTTED WITH RECOVERY.

Giant phagocytes	Total ear	Recovery within: 1 month	1 to 2	2 to 6	over 6	with mastoidectomy
Many	67	18 (27%)	7	6	6	30 (43%)
Moderate to rare	92	63 (67%)	11	8	—	10 (11%)
None	68	68 (100%)	—	—	—	—
Total	227	149 (65%)	18	14	6	40 (18%)

The table clearly shows that if no giant cells were present the recovery was not prolonged. It also shows a partial correlation of mastoidectomy and prolonged recovery with the presence of the giant phagocytes. It is largely true, therefore, that the more giant cells there are, the longer the recovery period. In order to explain the prompt recovery of 27 per cent of those with many phagocytes, some factors other than the bone infection are indicated, such as good resistance, in part, possibly due to the phagocytes themselves, adequate drainage, and other factors such as the penicillin which was used in five of these 18 cases.

In the table all gradations of severity of otitis are represented—from those that drain only a day or two, to some patients that were sick for years and in whom all kinds of complications occurred. Description of the details which help explain the cytology follows.

The 67 cases with many giant cells in at least one smear are of especial interest. Thirty of these had mastoidectomies. The typical pre-antibiotic purulent coalescent mastoiditis had many giant phagocytes as mentioned above without exception. These cases include the complications of zygomatic swelling and abscess, perilabyrinthitis, facial paralysis, and meningitis. When the discharge continued after the operation, giant cells were present as long as the infection remained active. The duration of the infection, if active, makes little difference in the number of phagocytes, because as many giant cells were present in an otitis of several weeks' duration as in one of two years and another of six years of continuous infection. On the other hand, as a case becomes "chronic", the phagocytes disappear.

The remaining 37 cases with many giant phagocytes did not have mastoidectomies. It seems likely that mastoidectomy would have shortened or lessened the disability of many of the 19 patients who took more than a month to recover. These had definite signs of mastoiditis. Several of the operated cases really belong in this group, because they were not operated upon for a long time, actually several years, after the recommendation for mastoidectomy had been made. Many of this group had remissions or temporary improvement which made diagnosis of surgical mastoiditis difficult. Nine of these received large amounts of antibiotics, but they were usually given in short courses until the mastoiditis was diagnosed.

Additional information is gained by studying smears taken on several occasions during the course of the otitis. The numbers are small but suggestive. Of the seven recovering within a month, six had at least one of three smears with few or no giant cells. Of the ten who took over one month to recover, only two had smears with few to no giant cells. It would seem that if an infection initially shows no giant cells, it may become only moderately severe. It would also seem that as an infection which has been showing many giant cells begins to recover, the giant cells will decrease in number.

The 68 cases with no large phagocytes present quite a different picture. All had dry ears within a month. None were very sick. There were five who had recurrences usually due to recurrent colds.

The large group of cases with a few cells, in general, lies between the two other groups in the severity and duration of the infection. Some of the cases had mastoiditis but not of the severe type seen with many giant cells. There were no major complications; nevertheless ten had mastoidectomies. The most severe was a case first seen after 15 months of earache and discharge. He had been treated with large doses of antibiotics most of the time but had flared up every time they had been stopped. In several, the severity of symptoms from an intervening external otitis forced the issue. Two were operated upon rather hastily at the end of three weeks and showed little pathology in the mastoid bone. A few giant cells were also found in some of the chronic cases. The finding of giant phagocytes, even in small numbers, gives an indication of more serious involvement than the finding of no large phagocytes, providing, of course, that fresh drainage from the middle ear and mastoid can be obtained.

TABLE 2. CASES OF CHRONIC MASTOIDITIS—33.

	in aural discharge	from mastoid cells
Many	0	2
Moderate to few	8	2
None	20	9

Table 2 gives a summary of the cases of chronic mastoiditis. It shows that large phagocytes are usually not found in smears from "chronic" aural discharge; bacteria are numerous and the leucocytes are very degenerate. The squamous epithelial cells are numerous and show a fatty type of degeneration. If a chronically infected ear becomes acutely infected, pain occurs, discharge increases and the cytology takes on the cells typical of acute infection. As indicated in the Table, there are two radical operations with many giant phagocytes.

One of these two cases again brings up the problem of acute versus chronic infection. He gave the history of ear trouble starting three years before with scarlet fever. He had had two simple mastoidectomies during the first year. His ear was still discharging and at the radical operation smears were taken which showed many giant phagocytes and the other cells typical of acute infection.

In the other case, fever, pain, and facial paralysis developed suddenly; the eardrum was intact, but the superior canal wall sagged with a large cholesteatoma. Pus from the mastoid showed many large phagocytes and also the fatty degenerated squamous cells.

This combination of acute and chronic cytological pictures is also found in the 13 cases of infected radical cavities. There were two cases in which the original acute otitis had never subsided in spite of treatment, and both simple and radical mastoidectomies. The other eleven were cases of acute infection of healed cavities. One case had three attacks of acute infection in her old cavity. Smears from two showed no large phagocytes, while smears from another more severe attack showed many.

DISCUSSION.

This cytological finding of phagocytes in aural discharge has proven to be an additional laboratory aid in the diagnosis of mastoiditis. Now with the use of antibiotics its chief importance may be in helping to differentiate acute otitis media from external otitis and from chronic otitis media and mastoiditis. In combinations of these diseases it may be helpful in the evaluation of the severity of each factor. This cytological information may be useful in deciding on the course of treatment and the extent of diagnostic procedures to use in a particular case, especially in view of the well known masking effect of antibiotics on mastoiditis.

The meaning of "acute" and "chronic" mastoiditis is clarified by the cytology of their exudates, as the two have quite distinct cytological patterns. The duration of the "acute" type has occasionally been found to be even as long

as several years; whereas, in other cases, chronicity has developed in a few months. The persistent finding of histiocytes in the smears of otitis media may be as important a factor in the diagnosis of acute surgical mastoiditis as is the prolonged duration of purulent discharge.

This type of pathological cytology, carefully studied, aids in evaluating the severity of the simple acute cases as well as helping to understand the complicated cases characterized by remissions and reinfections. All cases need to be evaluated on an individual basis. The smears of aural secretions, when adequate and available, with their various cellular patterns, may prove to be an accurate index of the pathological processes occurring in the tissues.

SUMMARY.

1. The phagocytic cells of aural exudates have been identified and presented as an important element of the cytology. They are easily seen with Wright's stain.

2. A study of the exudates of 277 cases of various types of otitis media and mastoiditis led to these conclusions: a. the large phagocytes are an indicator of relatively severe acute otitis media and usually mastoiditis; b. absence of the phagocytes in the discharge of acute otitis media indicates a relatively mild illness of short duration.

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THE FITTING OF HEARING AIDS AS AN OFFICE PROCEDURE.*

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Whenever an otologist has succeeded in convincing a patient that he should use a hearing aid, the patient will invariably and immediately ask two questions: (1) "which, in your opinion, is the best hearing aid?"; (2) "how should I go about getting one?" The first of these is easily answered with the categorical statement that there is no "best" hearing aid that would suit all cases equally well. For many years, the Council on Physical Medicine and Rehabilitation of the American Medical Association has assumed the responsibility of setting certain standards of construction and performance, and of testing the various makes and types of hearing aids on the market and new ones as they appear. The Council has done yeoman service to the otologist by thus relieving him of a task to which he could never do justice. A word of appreciation is due here to Mr. Howard Carter, the secretary to the Council, who for years has been in charge of this excellent program. Lists of "accepted" hearing aids are being published periodically in the literature and also in the form of reprints which may be obtained by applying to the Council. In the Transactions of the American Academy of Ophthalmology and Otolaryngology for November-December, 1950, is a list comprising no fewer than 29 makes and 69 models of wearable hearing aids. Undoubtedly this list has grown considerably during the past three years.

In consideration of the efficiency and integrity of the Council we must assume that all Council accepted hearing aids are "good". To determine, however, which aid is best

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for a particular patient is quite another matter. To state it bluntly: no hearing aid is better than its local agent. In the hands of a conscientious sales agent an instrument that just barely conforms to American Medical Association standards may do wonders for the user, whereas the most perfect instrument may be nothing but a source of constant trouble and expense, if it be handled by a "super salesman" who thinks of nothing but his profit.

The literature, both professional and commercial, is replete with suggestions and directions for the proper selection of hearing aids, but no single method seems to be universally applicable. To tell the patient nothing more than to get himself a hearing aid will not do. As Day[1] so aptly put it, an ophthalmologist might as well tell his patient to "try some eye glasses". Advice of such kind places the patient face to face with a situation that he is utterly incapable of handling. Being thus put on his own, he can do no more than study advertising matter. Not knowing what to expect of a hearing aid, he is totally unable to judge on his own. Consultation with other hearing aid users at random would confound him utterly, because ten users would probably recommend ten different instruments as being the "best". The end result of his endeavors would very likely be bitter disappointment, and one more specimen would be added to the huge collection of hearing aids tucked away in bureau drawers all over the country.

At the other end of the list of procedures would be the elaborate time and money consuming methods as employed in some of the Armed Services Hospitals.[2] Somewhere in between would be the "Hearing Clinic" as described by Carhart.[3] Books have been written for the laity[4] and for the profession[5] to guide us and our patients. Some of the advice given in the literature can be followed easily, but often it can not; thus, it is neither practical nor desirable that the patient "go shopping", trying each of several instruments for, say, a month each. By the time he has tried three or four, he will have forgotten how the first two performed, and he may finally decide to "wait a while".

Nothing could be more fatal to his program of rehabilitation. Incidentally, many agents refuse to sell hearing aids on trial, because such a system is unsatisfactory to all involved.

The problem is somewhat simpler in cities that enjoy guilds or societies for the hard of hearing. Every otologist should know the group nearest to him, and he should know what it is prepared to do. More than that, he should join such a group, if for no other reason than to give it his moral support; also, he should become a member of the "American Hearing Society" for the same reason. In *Hearing News*, the monthly publication of this society, he will find much that will aid him in helping his hard of hearing patients. *Hearing News* deserves mention here particularly because it lists local societies as well as teachers of lip reading. Thus, the issue for May, 1951, lists 116 local groups in as many cities and towns in the United States and Canada. The patient should be urged to join his local society. If it be large enough, it may offer him a hearing aid selection service that would put him under no obligation. The degree of efficacy of such service varies, of course, with the individual groups.

Several years ago it was suggested to the writer that he take over the task of selecting hearing aids for the patients of his colleagues. The technique employed, although no longer practiced, will be described in some detail because the writer learned much about the facts and fancies of hearing aids that he could not have learned any other way. Several instruments were obtained on loan from some of the local agencies who were given to understand at the outset that no favors were to be asked or expected. The selection procedure was as follows:

First the patient's ears were inspected as regards the appearance of the canals and drums. Then a careful hearing study was done with the pure-tone audiometer, tuning forks and, most important, the live voice both whispered and conversational. Tuning forks and audiometers may tell us what the patient can "hear" in terms of cycles and decibels, but only the voice test shows what he can understand. The writer

does not approve of the use of the recorded voice for this type of work, because it is the live voice with all its variations in volume and character that the patient wishes to hear the most. Every day he has to listen to many varieties of voices under conditions that are anything but "controlled". Whole sentences should be used for the reason that we are not in the habit of using words of only one syllable in holding a conversation. In composing test sentences, the patient's age and education must be given due consideration. Thus, for children of different ages special sentences should be available. In dealing with children, graded "Readers" will be found particularly useful, because, in reading from a Third Grade reader to a youngster going to the Fourth Grade, one is almost certain not to overtax the child's vocabulary. Having ready-made lists of sentences available relieves the examiner of the necessity of having to think of them on the spur of the moment, and much time is saved. Numbers should be avoided as much as possible, except in the case of elderly patients who have been hard of hearing for a long time. They often find it difficult to remember even short sentences long enough to repeat them. In such cases it is, of course, perfectly feasible to give them the sentence in short phrases and to let them repeat them the same way. Since a patient's ability to understand the spoken word can be judged only by having him repeat what was said to him, one should avoid questions that can be answered by "yes" or "no", for, obviously such an answer would not always tell us what the patient actually heard.

One of the most useful instruments for testing voice perception quantitatively as well as qualitatively is the old-fashioned speaking tube such as severely deafened persons formerly used for person-to-person conversation (Fig. 1.). When we say "old fashioned" we most certainly do not mean "useless", for the speaking tube is still an excellent hearing aid, although somewhat clumsy and conspicuous. It offers us a means for determining at once and within a very few moments how well the patient might be expected to do with a modern electric aid. If he can understand a faint whisper through the speaking tube, there is no question

about the efficacy of almost any hearing aid; if he cannot understand a faint whisper but does well with the conversational voice of low or moderate intensity, he would still be a good candidate. If, however, he cannot understand even a fairly loud voice, then no hearing aid will give him speech intelligibility; he must then be told, that he will have to rely on lip-reading.

Fig. 1. Speaking Tube.

The above tests completed, the receiver of a hearing aid is now applied to the patient's ear by means of a straight plastic nipple provided with a piece of pure gum tubing in order to assure a tight fit (see Fig. 2). The patient is instructed in the use of the volume control, and the examiner places himself at a distance greater than that at which the patient could have understood him with the unaided ear. The examiner keeps up a running conversation asking the patient to adjust the volume control until he thinks that he can hear everything that is said to him comfortably. The volume control is then let alone and perception tested with sentences of the type used in the hearing test. The examiner must, of course, be careful to keep his voice at an easy conversational level; he must withstand at all

times the temptation of raising it in case the patient should not respond promptly or correctly. If any sentence is not repeated perfectly or is repeated only partially, it is best to proceed to the next one, and to repeat the missed sentence later. If too much of an issue were made of a missed sentence the patient might immediately become prejudiced

Fig. 2. Straight Plastic Nipple to fit standard receiver.

against the aid. During this test it may become necessary to have the patient readjust the volume control. As he becomes more and more accustomed to the sound of the aid, and that usually requires but little time, he will almost invariably find that he can get along with less volume than he at first thought necessary. As a rule, the patient should not be allowed to watch the speaker's mouth. Elderly patients, however, and those of any age whose hearing loss is severe and of long standing, should be allowed to use their eyes as well as their ears. We must remember that everyone, hard of hearing or not, makes use of lip reading in every-day life.

When both examiner and patient are satisfied that the volume control is at the optimum level, the examiner should walk to various parts of the room, now and then turn his back to the patient, and deliberately change his voice level and quality, always noting the patient's response as regards both accuracy and promptness.

The choice between air and bone conduction receivers should present no difficulties. In the writer's opinion air conduction receivers should always be the first choice regardless of how good the patient's bone conduction may be, for the reason that the higher frequencies, say from 1024 cycles/sec. up, which are essential for good speech discrimination, are transmitted much better by air than by bone conduction receivers. Patients with discharging ears or wide open radical cavities, on the other hand, should use bone conduction, providing they have adequate residual hearing by the bone route. Some radical cavities, especially those resulting from endaural operations, lend themselves well to the use of ear molds so that such patients, too, can be given the benefit of air conduction. A dry perforation is no contraindication to the use of an ear mold. Usually the making of the ear mold may be left to the sales agent except in cases of dry perforations or radical cavities which should be carefully packed with deep cotton pledgets by the otologist.

Next we must decide in which ear the aid is to be used. In this we must naturally consider the amount and quality of the residual hearing of either ear. If the hearing loss is severe in both ears, it is usually necessary to use the aid in the better ear or in both simultaneously. If the hearing is the same in both ears, or nearly so, molds should be made for both ears and the aid worn alternately on both sides. In such a case the patient would usually favor one ear, but if that ear should become irritated by the mold he could always use the other ear. If one ear has good speech discrimination, say at three or four feet and the other at only one foot, the aid had better be worn in the poorer ear. The patient would then be able to understand voices from near by in his unaided "good" ear with little or no difficulty, while at the same time, his poor ear would get the benefit of the aid; in other words, he would then be hearing well with both ears. If he were to occlude his better ear with the receiver he would have to depend on the aid exclusively, and, to make matters worse, the poorer ear would be useless.

During the first years of practicing this method, three or four instruments were used in the manner described and a final selection made on what the writer considered valid criteria which were, in the order of importance, the accuracy and promptness of the patient's responses and the quality of the sound as judged by the patient. It soon became apparent that the patient's judgment of sound quality could not always be trusted. He would often claim that the examiner's voice sounded "more natural" with one aid than with another, whereupon he had to be told that, having been hard of hearing for a long time, he had long since forgotten what a "normal" voice was like. He had to be given to understand that to become accustomed to "new" sounds and sound qualities was an important part of the rehabilitation of anyone using a hearing aid for the first time. A bit of gentle bullying was at times necessary to acquaint him with this fact.

After having followed, for some time the technique as described above, the writer gradually came to the conclusion that his attempts at making a truly significant selection during the brief time span of two or three hours were essentially fallacious so far as most cases were concerned. It became apparent that there was not enough difference in the performance of most of the accepted hearing aids to justify the time and effort involved in trying to make a selection and change was in order. The technique of the procedure remained the same, except that the number of hearing aids was reduced to one, and its object was no longer an attempt at making a choice, but rather, a test and demonstration. The purpose of the test is to ascertain whether the patient will benefit by the use of an aid and how much. The demonstration prepares the patient for his coming adventure in hearing. We have found this new form of hearing aid consultation most satisfactory. It is simple and "down to earth". Instead of being put on his own, the patient naturally realizes that his problem is being handled with impartiality and understanding by his otologist rather than by a businessman exclusively. The agent, on the other hand, realizing that the otologist is taking an active interest

in the rehabilitation of his patient, will be doubly anxious to give satisfaction. Obviously, the hearing aid consultation as an office procedure has its drawbacks: it is time consuming and may not always fit into the office routine of a busy practitioner; again, the necessary instruments may not be available, due to lack of cooperation of the local agents or because there are no agents in the locality.

What can be done in such a case? To have the patient buy an instrument "over the counter" in a drug or department store would be most unsatisfactory. There is a finality about such a transaction that does not at all fit into the picture of rehabilitating a hard of hearing patient. The selling and buying of a hearing aid should not be the end but the very beginning of a lasting personal relationship between the agent and the patient. The proper operation of a hearing aid requires a certain amount of aptitude and much practice and experience. The patient has to supply his own skill, but the salesman can give him many valuable hints that will enable him to get the most out of his aid in the shortest possible time.

Which instrument should the patient buy? As was pointed out above, we must assume that all American Medical Association accepted hearing aids are "good". Rather than have the patient do his own "shopping" it is perfectly in order that the otologist, who should be familiar with whatever types may be available, and who should be personally acquainted with the respectivc agents, take it upon himself to instruct his patient to buy a particular make, to learn its operating technique, and to keep in touch with his otologist and his agent. Most important of all, he must be urged to *use* the aid constantly regardless of whatever difficulties may arise. Under no circumstances should he ever think of "giving up" until he has used the aid for at least six months. He must be made to understand that a defective ear with the most efficient hearing aid in the world can never be the equivalent of a normal ear. He must realize that the aid, being nothing but an *electric sound amplifer,* will amplify all sounds that come to it and

that, consequently, it will often be more of an annoyance than a help. This is particularly true in patients with perception deafness who are exceedingly sensitive to adventitious or "background" noises.

A hearing aid will often fail its user, but that does not in the least detract from its value. One of the writer's stock arguments is that a one-legged person can get around very well on a crutch, but that a crutch is not to be expected to do everything that a natural leg can do. Many patients fear that they will become "dependent" upon their aid, and some even "have been told" that the use of a hearing aid may result in a further loss of what little hearing they have. Such arguments and fears we may counter by pointing out that it is better to depend upon a hearing aid than upon a bad ear, and that the use of a hearing aid promotes listening, and that listening promotes hearing.

What otologist has not heard the remark "I think I'll wait a while"? The writer's stock reply to this is: "And what do you want to wait for? Do you want to wait until your hearing has become so poor that even a hearing aid will no longer help you?" At times it is even necessary to shame a stubborn patient into using an aid by pointing out to him that a hard of hearing person is all too often a nuisance to his family and friends. All those around him are forced to raise their voices and to keep repeating what they are trying to convey to him. These and similar arguments often make it necessary for the otologist to use a good deal of "salesmanship", but such is entirely legitimate. The otologist derives no personal gain from it, and the patient will benefit by it.

Incidentally, the necessity of taking up lip reading must be routinely impressed upon every hard of hearing person, whether or not he uses a hearing aid. Lip reading is the most fundamental item of any rehabilitation program.

After what has been said about aids and the understanding of speech, it may seem strange to state that persons

who by all standards of audiometry have no useful hearing, can also benefit greatly from a hearing aid as an adjunct to lip reading. Shortly after the writer had begun his work in hearing aid selection, he was consulted by a young man of twenty-two who was a well-rehabilitated graduate of a school for the deaf and supposedly an expert lip reader. The hearing study revealed that he had no useful residual hearing whatever. Over the speaking tube, he could not understand even the name of his home town. All sounds were nothing but vibrations to him. Obviously, no hearing aid could be expected to give him understanding of speech. Like most lip readers, he got along well with his family and friends, but when the writer spoke to him he invariably looked at his father who had to act as "interpreter." A hearing aid was then applied to one of his ears, the volume turned up fairly high, and almost immediately he was able to understand practically everything the writer said to him. Not only did the aid supply him with a certain amount of rhythm, it also enabled him, after a short period of use, to distinguish vowel sounds by their vibration patterns, and further, it rendered him aware of sounds emanating from a source invisible to him; in short, it gave him a contact with his surroundings that he had previously lacked. This case was but the first of several which derived great benefit from a hearing aid, although most of these patients had no hearing to aid. To repeat: persons whose hearing is either totally absent or insufficient for speech intelligibility, should be given hearing aids as soon as they have become thoroughly proficient in lip reading. In the speech training of young deaf children, the hearing aid has for some time been an invaluable tool. As to the choice of receivers, it may be argued that the bone conduction type should be ideal for such cases. In fact, some patients have been known to get along well with an ordinary bone conduction receiver held between thumb and finger. The writer, however, is convinced that air conduction receivers are to be preferred because the impact of powerful sound waves upon an ear drum would be more effective than the rather coarse vibrations of a plastic or metal plate against the thick skin over the mastoid area.

One more word about the "best" hearing aid. After a patient has been using, for about a year, whichever hearing aid he was told to buy, he, better than anyone else, will be able to pass judgment on its performance and usefulness as compared to others—he will truly be the expert.

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MEDICAL PROBLEMS.*

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(by invitation)
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I know in the program I have been given a topic called "Medical Problems". That should certainly give me enough territory to take up a few minutes.

I think we might divide our problems into different groups. There are certain medical problems; certain problems concerning the doctors and the public; certain problems concerning the doctor and the American Medical Association; and certain international medical problems.

I will just say a few words about each of them. First as to our immediate medical problems, I think we may say without fear of contradiction that American Medicine in our American medical care system is on a higher plane than anywhere else in the world. Nevertheless, I do not think we should be smug about it and sit back and feel perfectly satisfied to do nothing; because if we are to maintain American Medicine in the leadership and if we are to maintain it under a system of free enterprise, then there are certain things which we must do and there are several items which come under that heading. First of all, we need a better distribution of physicians than we have in the United States. We hear a great deal about our needing thousands more doctors. Well, the facts are that we have now in the United States more doctors in proportion to population by 25 per cent than any other country in the world. We have one doctor to every 750 persons. Our nearest competitor is Great Britain where they have one to a thousand, but unfortunately that one to 750 does not apply to the whole country. For ex-

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ample, in New York City there is one to every 450 persons, which I think any reasonable minded individual will say is too many. In some of our rural areas in the South we have one to 1800, which I think again anyone will admit is too few. How are we going to get better distribution? We are certainly not going to do it by increasing the number of doctors. You can double or triple the number we have and you still would have the same trouble in distribution because they would congregate in cities and stay away from the rural areas. There is only one way it can be accomplished, that is, by providing the facilities in rural areas to attract the doctor. Some of the Midwestern States have found that to be the case and the communities which were losing their old general practitioners, who were dying off and not being replaced by younger men, set up facilities to attract the doctors so that they could practice modern medicine. It might be a small hospital in some area or merely an office with a laboratory attached, including certain limited X-ray facilities and then it would be rented or sold to the doctor over a period of years through an amortization program. When they did that they had no trouble whatever in getting doctors to settle in these communities; but no doctor who has spent anywhere from ten to thirteen years of his life is going to settle in an area where he would have to practice medicine of the type that was in vogue 75 or even 50 years ago and personally I think the community would be better off without him if he would.

As to an increase in the number of doctors, they are now increasing at a rate faster than the population is increasing. Our medical schools are at an all time high, not only in graduating classes but in their entering classes. There are more people now entering and graduating from our schools than at any time in the history of the country, and that does not take into account the new medical schools which are in the process of organization, some of which are already under way. So that I believe that we shall get our increased number of doctors which we do need in an orderly fashion without any radical change.

There is another gap in our program and that is the individual suffering from chronic and degenerative disease. The number of those people is increasing because they have had good medical care. In other words, our life span has increased about 21 years in the last half century, so there are millions more people living to middle and old age where they are subject to these diseases than was the case a half century ago; and that problem is going to continue until we find some way of either preventing or curing chronic and degenerative disease. When we do I suppose we will all live forever.

I think one factor, while it has not anything particularly to do with the development of chronic disease, certainly is a factor in the death rate and that is what I consider a very stupid economic method we have in putting everybody on the shelf at the age of 65. In my personal opinion that retirement has killed more people than it has saved lives, except in very occasional instances. It is said to be all right to retire if you have a hobby. Well, certainly the individual who has been active all his life and then suddenly quits and does not have any other interest in the world to take up his time is not going to last very long. At least that has been my experience.

Another problem we have is to see that the indigent have a proper medical care program in the community. Many states have settled this in a very satisfactory fashion by cooperation between the welfare authorities or whoever handles it for the community and the local medical society and there is no reason why it cannot be worked out in all areas in the same way. I think we need a little wider coverage in our public health facilities. I believe everyone agrees that this is essential.

There have been numerous arguments and sometimes medical societies have been loath to approve local public health units which I think is a mistake. The argument, of course, has always been where public health stops and where private medicine begins. Certainly there can be no argument

as to environmental sanitation, protection of food, milk, water supplies, eradication of disease-bearing insects, prevention of communicable diseases and vital statistics. Those things have to be carried out on a community basis and cannot be done on an individual basis, and oftentimes they require what we might term a police power which the individual physician does not have.

I think we need an expansion of our voluntary medical care insurance system. Prepaid medical care is here to stay. It is just a question whether you are going to have it on a voluntary basis or compulsory basis. The voluntary programs have had tremendous growth. Insurance people have said it is the fastest growing insurance program in the history of insurance in this country, which is a pretty insurance minded country. Nevertheless, it is not yet the whole answer, because it has certain gaps in it which must be eradicated. Millions are being added to the rolls each year. In hospital insurance, for example, we now have practically 90 million people covered. In insurance against surgery and maternity there are about 66 million covered, and for medical care in whole or part there are about 30 million; but there are two gaps, one of which is rapidly being closed and that is protection of those over age 65. When these plans started they were experimental and the programs, had they taken in those with chronic disease or those over 65, would have been broken financially very quickly. They could not do it. Now on a sounder financial basis they are expanding the coverage. Once the person is insured they are keeping him insured as long as he lives. That is rapidly taking place all over the country in the various types of plans.

The other gap is protection against long duration illness, what sometimes has been called financially catastrophic illness. Beginnings have been made on that, yet progress has not been wholly satisfactory.

One other thing in which I think the relation of the doctor and the public is important is the establishment of com-

munity health councils. Medicine is no longer a matter of just doctor-patient relationship in the scientific aspects of medicine but it involves a lot of other things and there are many individuals and many organizations in the community which have a stake in health. A community health council can be formed from among these various organizations of individuals but in my opinion the community health council will not be successful nor will it be guided in the right direction unless the medical profession takes the lead. There is no one who has quite the qualifications of the doctor to give leadership in health matters, and I think it is up to him to do it, because if he does not somebody else will and the results may not be as satisfactory.

I believe all these changes can be carried out within our American system of medicine and our American way of life. I don't think that we have to consider, and much less adopt, any of the programs which have been in vogue in most other countries in the world.

I have had an opportunity during the last six years to go to Europe 12 times. Last fall I went around the world and I have seen these systems in most every country, and although I don't wish to criticize any country for the system which it adopts—that is its own business and what may work in one country may not work in another—nevertheless I say without any reservation whatever that I have seen no system in any country that I want to see adopted over here. Every one of the systems which is government controlled or government sponsored is operating in the red. There isn't a single one of them without a deficit, and they try to remove that deficit by restricting benefits to the patient, restricting medication and treatments by the doctor and, of course, reducing remuneration to the doctor.

The Japanese have two government controlled systems: one for industrial workers, one for the farm workers, and the doctor who takes care of those patients, if he examines a patient, he gets the munificent sum of 20 cents; if he reexamines him he does not get anything. The surgeon fares

pretty well because if he takes out an appendix he gets $7.50! In Greece the doctor who might receive $10 for a consultation in a private case, in a government case would receive 50 cents.

Well, what the doctor gets is not the important thing. It is the fact that with such a type of plan you get assembly line medicine and the patient is the one who gets the short end of the stick; so, as I said, I have seen no system anywhere that I want to see adopted over here.

Socialized medicine in the United States I think is no longer an immediate danger, but whether it becomes a danger again depends a great deal on the medical profession. I think if we remove the gaps in our present system and improve the distribution of medical care along the ways in which I have outlined, then I don't think we ever need to fear it again; but if we don't, then it again will rear its ugly head. So much for the domestic level.

There are one or two things which I believe the doctors can help a great deal. They must take an interest in community affairs. I feel the time has long passed when the doctor can confine himself purely to scientific medicine because there are so many other factors now pertaining to the profession of medical care that we cannot close our eyes or bury our head in the sand. The doctors must interest themselves in the economic and social aspects of medicine because they are very intimately connected with its distribution. I hate the term "social medicine" because everybody thinks you are talking about socialized medicine whereas, as a matter of fact, they are quite different.

I was at the American College of Physicians the night before last and I said there was no more relationship between social medicine and socialized medicine than there was between ophthalmology and proctology, and I thought maybe it was a pretty good analogy. Nevertheless social medicine pertains to the entire environment of patients, their housing, their clothing, their nutrition, their economic level and so on. Those things must be taken into consid-

eration. It is rather unfortunate I believe when medical societies advertise a meeting on socio-economic aspects of medicine and very few attend. That is going to be our undoing unless doctors do take an interest in these aspects of medicine. Our county societies used to be the most influential bodies in health that there were in the country. Everybody looked to the county society for advice and leadership but because of the lack of interest which has developed recently, perhaps due to over-organization in medicine and too many meetings to go to, attendance at county societies has fallen off and the result is the county society has lost the position of leadership which it once had and it must be restored.

I said there was no particular danger at the moment of socialized medicine in the United States from the domestic level. There is a certain danger I think from the international level, and as you know, there are more and more problems being discussed and decided on an international level pertaining to health and medicine than at any time heretofore.

One of these organizations which is concerning itself very directly with medical care, particularly under Social Security, is the International Labor Organization. A year ago this International Labor Organization held a convention in which they brought in a report recommending compulsory health insurance or government controlled insurance for every country in the world adhering to the International Labor Organization, and the United States has been a member of it since 1933. What is not generally realized is that all that would be necessary to put that program into effect in this country would be for the United States Senate to approve it, just as it would a treaty and you would have it. The House of Representatives would have nothing whatever to say about it. This whole program was highlighted in the Journal of the American Medical Association, May 31, 1952, and also later on in the proceedings of the House of Delegates and in an article written by one of the representatives of the United States to this International Labor Convention, the

one who represented management. If you have not looked these articles over, I suggest you do so, because it shows you that again we cannot keep our heads in the sand just because things are the way we like them in this country and not pay attention to what is going on abroad.

There has been developed a World Medical Association which is an organization of the National Medical Associations of the world and it is the only organization, aside from the purely scientific ones, which deal only with scientific matters, which can speak for the practicing physician. All these other organizations discussing these problems are governmental in origin and governmental in viewpoint. While government has a certain part to play, and that we cannot deny, in my opinion it should stay in its place and not step over into the field of medical care.

I want to distinguish the World Medical Association from the World Health Organization because they are confused in most people's minds. The World Health Organization is a branch of the United Nations and it represents the governments of the world in the field of health, more particularly within the field of public health, whereas the World Medical Association is an organization of National Medical Associations and represents those associations and the practicing physician, and that association is the only one that gives you a voice in discussing problems at an international level so far as medicine is concerned. It has been rather astonishing to me in going around the world and attending the meetings of the World Medical Association to find that medical problems are pretty much the same the world over, particularly the social and economic problems. They may differ in degree and they do, of course, but they are the same problems basically. What perhaps is more astonishing is that the doctors of the world, no matter where they come from, think pretty much alike about it. I recall in Geneva, in 1948, the World Medical Association went on record as setting up certain principles pertaining to medical care if it were to be administered under Social Security. There were 12 of these principles and the delegates which

were there at that time from 31 nations, argued for a day and a half over those 12 principles but most of the argument was on matters of language. As soon as the language was clarified those 12 principles were adopted unanimously, which I think in itself is an instance that doctors think pretty much alike when you can get them from 31 nations to agree on 12 items of medical care; because in dealing in three languages, English, French and Spanish you have to be very careful in translating, and those of you familiar with more than one language know you cannot translate words because the same words mean something quite different in another language. You must translate the idea. When you put over the idea then the other person understands you.

The World Medical Association has only one member per country, that is, the National Association, so the American member is the American Medical Association. We have, however, in the United States, a United States Committee of the World Medical Association in which individual membership is possible and you have all the privileges of membership except actual voting, including getting all the publications, attending the meetings, etc., and making contacts for you of medical facilities abroad. Of course the only ones who can vote are the delegates of the National Associations. There are now 43 nations in the Association and several more probably will be in during the next year. So much for the World Medical Association.

As to medical education, as you know, our medical schools are in a little difficulty financially and it is a question of how they are going to be supported. Are they going to be supported by private enterprise or are they going to be supported by the Federal Government? Some people say, "Well, why not let the Federal Government do it? They probably would not try to control the schools and they are the only ones who have got any money anyway". I disagree on both counts. First of all, the Federal Government does not have any money that it does not take out of your pocket and mine. In the second place, I do not feel

assured that the government would not try to run the medical schools. I have said this before and you may have heard me say it, that a year ago the Education Division of the then Federal Security Agency reprimanded a teacher who had been sent abroad under an international teacher exchange program by the Division of Education because when he got home he expressed himself rather feelingly on the subject of socialism as he had seen it in another country that he had visited. He was informed by the government agency that the publicity given his remarks might result in his whole area being excluded hereafter from the international teacher exchange program. So there is a danger.

Also you might be interested that in studies which the World Medical Association has conducted on an international level as to medical education, in the totalitarian countries where the government has an absolute stranglehold on the medical schools, one of the subjects taught is political ideology. Now I hope that the doctors realize the importance of keeping medical education free and unhampered in this country and I don't know whether they stopped to realize it before, but I think they all owe a debt to their medical schools because not one of you ever paid for your medical education. You paid your tuition. That probably represented not more than one-third of the cost of the school for educating you. The rest came out of endowments. Endowments are shrinking, and the investments from endowments are not so good as they used to be because of the high tax rate and consequently the schools are having difficulty in making both ends meet.

There are various ways in which you can repay your debt to the school. You can contribute direct to the school or contribute to the American Medical Education Foundation. If you do that the latter will be distributed to all the schools, if you don't earmark it; if you want it to go to an individual school you can so earmark it and that school will get your entire contribution and the school will be informed that you have contributed so much to its operating fund. All funds

contributed to the Medical Education Foundation go to the schools; not a single cent is deducted for administrative overhead. That is all paid by the American Medical Association independently, so every cent that you contribute will go either to schools in general or to a specific school if you wish.

The doctors have been doing pretty well. In fact, there is another organization known as the Medical Fund for Medical Education which has its headquarters in New York, and of which the Hon. Herbert Hoover is honorary president. We have been working in close collaboration with them. They are going after funds from industry. The Medical Education Foundation is seeking funds from doctors and medical societies, but the two funds are combined when they are distributed so the schools get one check, but each school knows who gave what. I think it is very important that we see that our schools are adequately supported because once the government gets in, bear in mind that the private contributions, which are no small amount, that the schools still receive, even though they are not adequate, will dry up because everyone would say, "The Government is doing it. Why should we contribute?" Should the government reduce or stop contribution, which it might well do in case of a depression or total war, the schools would be worse off than they are now.

I would like to say just a few words about the American Medical Association. I think perhaps the American Medical Association is the most maligned organization that there is in the United States and most of the criticisms are based on lack of knowledge. I have been somewhat shocked to go around the country, as I have, and find how few doctors really know anything about the American Medical Association. During the past two years as President-Elect and President I have had, I suppose, at least 2,000 letters sent to me personally which I had to answer. I would say that half of them were based on lack of information about the American Medical Association. The American Medical Asso-

ciation is a large organization and in an organization as large as that you don't find everybody agreeing, but difference of opinion is healthy. That is what makes the world go round. If we did not have difference of opinion I don't think there would be any sense having an association. If everybody thought alike on the programs there would be no need to do anything. There would be no need for organization. We have to bear in mind that in any organization the majority has to rule until the minority can convince the majority that they are wrong and remember this, that no organization is ever going to pursue or adopt any policies which are at variance with the wishes of its members. It would be suicidal if it did.

Again I find that doctors have no knowledge of what privileges they have as members of the American Medical Association. Of course, in your county society you know you have a vote on everything. Your county society elects delegates to the State and those delegates in turn elect delegates to the American Medical Association. The House of Delegates of the American Medical Association is the policy-making body. It consists of the State delegates and one from each scientific section. No officers of the Association or Trustees have any vote whatsoever. No policy, no report, no resolution is adopted by the House of Delegates when it is introduced without its being referred to a reference committee, just as legislation is handled in Congress. When anyone introduces a bill in Congress it is referred to a committee. These reference committees hold hearings just as congressional committees hold hearings but one does not generally realize that any member of the American Medical Association, no matter whether he has ever been a member of the House of Delegates or not is entitled to go before that reference committee and express his opinion and argue for or against the adoption of any item and in that way I think it is even one step better than Congress because Congress does not always hold hearings and sometimes when it does it limits the time you can have and limits the number of persons who can speak. That

is not true in the American Medical Association. I do not know how you could have anything much more democratic than that.

Then we hear a lot about the American Medical Association being a hierarchy of a few individuals. Well, I think perhaps I can scotch that, if you will pardon a personal reference. I was elected to the Board of Trustees of the American Medical Association in 1944. When I was named President-Elect in 1951, seven years later, there was not a single man on the Board of Trustees who was there when I went on. You don't have a hierarchy when you have a complete turnover of managing personnel in seven years.

I think perhaps the greatest mistake the American Medical Association ever made was that it did not charge dues from its very inception because you never appreciate what you get for nothing and people have taken the American Medical Association for granted so long, that they don't stop to consider what they owe to it. I have heard doctors say, "Why should I support the American Medical Association? What did it ever do for me?"

If you stop to think about it, the fact that medical education in this country is unexcelled anywhere in the world and that this country has become the Mecca for medical training, whereas it used to be Europe, is due entirely to the American Medical Association; because it was the American Medical Association that started the campaign to eliminate second and third rate medical schools and the diploma mills, which were operating in this country half a century ago. A lot of rocks were thrown at it at that time by some of those sponsoring these proprietary schools but I think everybody now realizes it was a great thing for medical education; so the fact that you graduated from a first class medical school you owe to the American Medical Association.

A half century ago there were no criteria by which any doctor or any citizen could evaluate the efficacy of any drug or any therapeutic agent. No doctor could evaluate the efficacy of any apparatus used in diagnosis or treatment but

through the Council on Pharmacy and Chemistry of the American Medical Association and the Council on Foods and Nutrition, the Council on Physical Medicine and Rehabilitation, etc., standards have been set up which must be met in order to receive the seal of approval. Certain levels must be met. You know if a drug or other therapeutic agent has the seal of approval of the Council of Pharmacy and Chemistry, for example, that that drug must have had sound pharmacological and clinical evidence submitted about it before it was approved. You know that there have been no unwarranted claims about it, no unwarranted advertising. You know that the formula is available on the bottle. It is not a secret remedy. You know that if there are any dangers connected with it the dangers are clearly printed, so you have something on which you can hang your hat. That is something that no other medical association in the world has done for either the public or its members.

The standards in industrial health and in rural health have very largely increased in this country through our Councils on Industrial and Rural Health.

The Council on Medical Service has been concerned with the economic problems in medicine, insurance, care of the indigent, federal medical services and so forth.

The Bureau of Investigation has done a tremendous amount in showing up quacks, extraordinary claims, charlatanism and fraud. The Bureau of Investigation has over half a million cards in its files giving information about various quacks and quack remedies which have been foisted on the public. As the result of the information which has been published by that Bureau in the Journal and in other publications the Association has been sued for I don't know how many millions of dollars by these quacks and charlatans and the producers of these patent frauds, but up to date of all the millions it has been sued for, one outfit collected one cent and one collected a dollar because of some technicality in the charges, so it proves pretty well that what we have had to say about these groups was true.

The Federal Trade Commission has used a great deal of the information in prosecuting these various individuals and organizations. I feel that every doctor owes the entire environment in which he practices medicine today to the American Medical Association, and I hope that I have succeeded in telling you a little about it that you did not know before and that you realize that the American Medical Association is concerned only with one thing and that is in bettering the distribution of medical care and protecting the practice of medicine from government interference. If you do not like what your Association does, get in and do something about it. I hate to see a person stand on the side and criticize and then not lift his finger to do anything to correct what he considers to be wrong. As I said before, no association has ever adopted or adhered to any policy which is at variance with the will of the members. If you do not get in and take part in your local societies, the state society, then it is your fault if they do things of which you do not approve, because the doctors have a powerful voice in the management of the affairs, not only of this Association but of this country. They are one of the most influential groups; because nearly everybody passes through a doctor's office sooner or later and he wields a tremendous influence and it is up to you to wield that influence for good.

THE ROLE OF THE RHINOLOGIST IN RHINOPLASTY.*†

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The role of the rhinologist in rhinoplasty is a fourfold one: first, he is a student; he should familiarize himself with the subject and devote himself to the necessary studies in a manner which is diligent and receptive; second, as an investigator he must be a careful observer of his own work, keeping complete records and bring to his investigations a broad scientific viewpoint based upon a background of general medicine and surgery; third, he must teach to develop the fullest appreciation and perception of the subject; fourth, he should review the whole field, eventually, in the role of a critic.

The evolution of the rhinologist in rhinoplasty is not a quick or short step. Given that he is well trained, is perhaps certified by the Board of Otolaryngology, that he has had some years of experience in the specialty, I venture to say that this evolution is a process that bears the fruits of real accomplishment in not less than five years.

The student must reorient himself and assess his own experiences and attitudes in the light of what fifty years of septum and sinus surgery have and have not accomplished and what fifty years of rhinoplasty have and have not contributed to the knowledge and armamentarium of the rhinologist. Perhaps a quick way to obtain a fair perspective will be to look at a few typical patients who come into the consultation rooms of rhinologists:

Case 1: This man was a professional boxer. He is 35 years old. His nose was operated upon twice. Each time a submucous resection

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of the nasal septum was performed. His present complaint is difficulty in breathing and no improvement following the two previous operations. His characteristic flattened boxer's nose more than suggests that reconstruction of the external nasal pyramid is indicated.

Case 2: This young man of 24 was also operated upon twice, but this time, the operations were rhinoplasties. The present complaint again is difficulty in breathing and persistent deformity not relieved by the two previous operations, and a study of the patient and the photographs show the definite evidences of uncorrected extensive nasal septum deformity.

Case 3: A young lady aged 30. Again the patient was operated on twice—first a submucous resection was performed and later a rhinoplasty. Her complaint is also difficulty in breathing. She has a dislocation of the caudal end of the septum which was not corrected by either previous operation.

Case 4: Man aged 53. It is seen that his nose leans markedly to the right. A submucous resection was done about 15 years ago, which not only did not help him, but actually increased his distressing symptoms, which were difficulty in breathing, nasal discharge and headache; also, three years after his operation, there gradually developed a septal perforation, which at this time is more than a centimeter and a half in its smallest diameter.

Case 5: This lady of 29 had a submucous resection operation of the nasal septum which was followed very promptly by a saddling of the dorsum of the cartilaginous vault and which gradually became worse. At the present time a marked deformity can be seen.

Case 6: This young woman of 28 with symtoms of difficulty in breathing accompanied by a chronic nasal discharge not relieved by the usual medical methods, had a submucous resection operation. Her symptoms were, in general, aggravated, and the incidence of colds increased very markedly. The picture of the base of her nose indicates the inadequacy of the nasal projection.

Case 7: Woman of 50. Ten years ago had a septal abscess following a very virulent ethmoid infection. After drainage was instituted and healing occurred, a characteristic deformity of the pyramid was evident, pathognomonic of absorbtion of the septum cartilage together with the upper lateral cartilages.

Case 8: Boy of 10 had continuous epistaxis for two weeks. The anterior portion of the septum was soft and boggy. It was opened, and a hematoma was diagnosed and evacuated. Further inspection and probing showed abscence of some of the septal cartilage and necrosis of much of the remainder. The need for septum repair to prevent vault and lobule deformities is apparent.

Case 9: Young man of 20 has a markedly twisted nose and complains of nasal obstruction. This external deformity is due entirely to the distortion of the septum.

Case 10: This girl of 16 was in an automobile accident, and her nose was fractured to the right. The nose was straightened but six weeks later the deformity recurred. The fractures—dislocations of the septum require concomitant treatment.

This very brief review is enough to show that many common nasal problems revolving around the septum cannot be solved simply by submucous resections but require procedures to correct defects of the inside of the nose and of the external nasal pyramid at the same time, or in related though separated operations. A familiarity with the many procedures involved in both extensive septal surgery and external nasal pyramid surgery is imperative for all of those who wish to work in any part of this field. The man who works on the septum must be familiar with the work to be done on the external nasal pyramid. The man who works on the external nasal pyramid—the man who does rhinoplasty—should be thoroughly acquainted with the problems of the nasal septum and especially its relationship to the rest of the interior of the nose from a medical as well as a surgical point of view.

The accent on the septum and its functional association with all its neighboring structures gives the investigator a stimulating interest in a fertile field of major importance. He sees that a more basic and broad statement of the functions of the nose needs to be made. He notes that the nose is more than an air warming and moistening apparatus. He knows that the nose cleanses the inspired air but not how this is accomplished. He sees that the manner in which the dust particles are deposited on the mucous membrane is not just a matter of simple contact but is influenced a great deal by the air currents. The deposition of dust particles in a precise locality is important to nasal comfort. He wants to know more concerning the air currents as they pass through the nose—such as the regulation of their shape, their size and velocity, their turbulence (and what creates it) their direction, and their pressures. He again learns how the afferent nerves of the nasal thoracic reflexes are stimulated by these air currents, and in this connection sees the need of further study of the anatomy and the physiology of the vestibule, the lobule and the upper lateral cartilages. He wants to know more about the variations of the space between the nasal dorsum and the anterior ends of the turbinates, the width of the nose and the relationship of the lateral

walls of the nose to the septum. He is struck by the realization that the hard septum actually creates two similar and complete noses. He wants to know, therefore, why the human requires two noses. In surgery of the septum he has been dealing with a party-wall made of hard bone and cartilage and concludes that this must not be replaced by a partition made only of "mucosal wall paper."

Shall these and the many allied topics and problems be the concern of the rhinologist? Hilger in the May-June issue of the Transaction of the Academy of Ophthalmology and Otolaryngology, 1952, writes: "the problem of architectural nasal obstruction has largely resolved itself into the proper surgical correction of the anterior third of the nose and septum. Rhinoplastic surgery has a physiologic as well as a cosmetic responsibility—The basic science of the region and the problems of surgical technique are those best known to the rhinologist."

The rhinologist in this country at least has accepted the responsibility of progress in nasal health, and during the last decade, slowly but surely has aroused a great concern and enthusiasm which have resulted in many significant advances, changes and contributions to the science.

1. To begin with, he has started out with basic anatomical problems. Through clinical, anatomical and photographic studies of humans of all ages and comparative studies of the lower animals, such as the horse and the cow, a better, clearer knowledge of the structure of the base of the nose has evolved. The medial crura, the lateral crura, extending into the space of the vestibule reveal their function as barriers or baffles to the air streams. The cul-de-sac which is very marked in horses, for example, is more than indicated in the human and subserves a useful function.

2. He is impressed with the effects of trauma on the growth of the young nose. Birth injuries are common and the apparently slight injuries in childhood may seriously impair the quantitative and/or the qualitative development of any or all parts of the pyramid.

3. The relationship of the upper lateral cartilage to the septum forming the valve of the nose is common knowledge, but the detailed anatomy of the upper lateral cartilages is not so well known. The terminal portion of each upper lateral cartilage usually is not attached to the septum. As one goes proximally, however, the attachment becomes firm and the angle at which it is attached changes. This, of course, is of great importance when one contemplates the shortening of the nose to include the shortening of the upper lateral cartilage. Can one make the proximal third of an upper lateral attachment take over the function of what is normally the specific function of a specialized terminal third?

From a physiological standpoint the rhinologist versed in rhinoplasty thinks of septum deformities on a functional basis. He also divides them into deviations and obstructions, but adds impactions and septums producing tension. Included among the latter are practically straight septums which may produce serious functional disturbances. As examples there are noses which are high and thin, and long noses in which the hard septum encroaches upon the membranous septum and the columella. Excessively shortened though straight septums may also produce symptoms of tension.

He has become aware of an important concept in respiration, namely that he must not limit himself to making a diagnosis only of a good airway, or of a good breathing space; he must determine whether or not there exists sufficient *resistance* in the upper respiratory tract, especially the nose, to both inspiratory as well as expiratory air currents.

It has been an illuminating experience to see the part the nose plays in the physiology of sleep and bodily rest. Of especial interest to the rhinologic surgeon is the influence here of the septum, the turbinates and the upper lateral valves, and he has added this information to his growing list of functional tests of nasal efficiency.

The rhinologist has become alerted to and recognizes the psychogenic stresses in the production of severe vasomotor disturbances and the psychological and psychotic disturbances of patients who may require or request septal and external pyramid surgery.

The rhinologist in his investigative efforts has been able to make several contributions to the catalogue of nasal diseases.

1. Atrophic rhinitis has been extended to a larger grouping, nasal atrophy, which includes not only involvement of the mucous membranes but also of all the structures of the nasal pyramid.

2. A common illness seen in many children as well as in adults producing severe intranasal symptoms is a condition designated as ballooning of the upper lateral cartilages. This is contradistinguished from collapsing of the upper lateral cartilages, which in the past has been confused with and sometimes amalgamated into the general term "collapsing of the alae".

3. Frequently associated with this is the problem of the large round or square tip which influences the functioning of the upper lateral cartilages.

4. The stretching of the upper lateral cartilages by an excessively high septum produces a narrowing of the angulation between the septum and the upper lateral cartilages, (the valve) which in turn predisposes to a collapse of the upper lateral cartilages, on inspiration.

5. The rhinologist has also described the somato-psychic effects of nasal disease.

6. Insufficiency of the nasal roof (the dorsum) is a less known syndrome suggested by the rhinologist. This consists of signs of nasal irritation aggravated by weather changes and associated with pain, usually referred to the

supra orbital and infra orbital regions. In this connection the X-ray study of nasal bones and their relationship to the frontal spines is emphasized. Congenital absence of nasal bones as well as the defects produced by excessive "hump removal" in so-called typical rhinopolasties cause this syndrome.

7. The rhinologist has a revived interest in the relationship between nasal function and lung function and between nasal diseases and lung diseases other than those associated with sinus infections. Through his investigations in combining surgery of the septum with that of the nasal pyramid he has again learned that he may be of service in the treatment of chronic non-specific lung ailments of a non-specific nature.

Technically, I suppose the greatest single thing which the rhinologist has learned and taught in this work is that a rhinoplasty is not a hard and fast routine thing. On the contrary, he has pointed out that there are many rhinoplastic procedures, and that they may be juggled about and used in many arrangements to help him design the best operation suited for the patient. In the hands of a rhinologist, an operation may start with a septal incision, or an intercartilaginous incision, or an incision at the caudal border of the lobular cartilage. There may be no intercartilaginous incision at all, and still be completely effective. He has made other contributions in concepts. For example, the shortening of the nose as originally indicated by Joseph, consists primarily of the removal of a triangular piece of cartilage at the caudal end of the septum. For the rhinologist, it may consist of a rotation of the lobule with a recession of the whole nose including the nasal spine. The rhinologist has been able to make advances in the moving and changing of the bony portion of the external nasal pyramid and has made it safer to do very extensive work in this area even when it is combined with complete reorganization or total removal of the nasal septum. Because of his familiarity with rhinoplastic procedures, the rhino-

logist has been able to develop more extensive surgery in atropic rhinitis and ozena. These include the moving of the lateral mucosal walls of the nose together with the inferior turbinates towards the midline and implanting between them and the lateral bony wall a variety of substances. Bone grafting here has been quite successful. He has removed the nasal septum in these conditions and substituted large cancellous bone grafts, which grow in the septal space and help provide the much needed increased blood supply for the mucous membrane which it supports and also an increased resistance and obstruction to the expired air which is so important for symptomatic relief.

He is developing many surgical methods which will help in the closing of large perforations of the nasal septum. He has developed the submucous exposing and surgery of the upper lateral cartilages. It is feasible to mobilize the nasal mucous membrane as it goes under the cartilaginous vault over to the lateral wall of the nose. The attachment of the mucous membrane here, in the angle of the valve, is a very tight and firm attachment, and only with meticulous technique can this be freed without tearing the mucous membrane.

Another application of the knowledge gained by knowing both the surgery of the septum and the surgery of the external nasal pyramid is the facility with which many operations can be performed with preservation of much of the blood supply. Operating through a septal incision for example without making additional vestibular incisions provides enough exposure for many pyramid corrections. The alar-facial incision for a lateral osteotomy can take the place of an incision in the vestibule or the elongation of an intercartilaginous incision.

The rhinologist has contributed the conception of eliminating a hump without removing any tissue. This consists of producing a downward posterior fracture in a manner similar to doing an infracture or outfracture of the bone

bridge. The down fracture actually pushes the hump into the nose without the removal of any portion of its substance.

The elimination and restriction of vestibular incisions, as often practiced by the rhinologist, contributes greatly to the prevention of stenosis of the internal nasal ostium. The separation of the nasal bones can be done intraseptally, and in this way the reflection of the nasal mucous membrane as it passes from the septum to the nasal bone and along the lateral wall remains intact. This can be a technical advantage in the operation, but more important it avoids a cut into the mucous membrane and so helps to preserve its function.

To conclude these remarks on investigative work one admits gratefully that the rhinologist has learned much from the plastic surgeon, but at the same time the latter can turn to the rhinologist for co-operation and guidance in the development of rhinoplastic procedures best designed for the preservation and restoration of nasal function.

As the rhinologist will make an effort to teach what he knows to his students, interns and residents, his colleagues in the profession and in the specialty, he will be stimulated to a better understanding of his own many problems. In the teaching he will give great evidence of his desire to share his knowledge and will in return stimulate the same feeling from those he is teaching and will get from them many important contributions to his own fund of knowledge. It is vital to note that one cannot convey the full essence of rhinoplastic procedures in a week, a month, or a six month course. This is a subject which requires a constant effort over a period of several years and an opportunity must be provided for the students to receive regular though intermittent instruction. The student must continuously replenish his interest and his fervor by talking and studying with his teachers and his fellow workers. To that end, I believe that short courses at frequent intervals are the answer. The teacher who starts giving this

sort of training has the obligation to provide these repeating periods of further instruction. When enough men are trained, I suppose that a study group on a national scale will evolve for this purpose.

After several years of an active program along the lines indicated, I think that the rhinologist must take on the role of a critic—not to be criticizing, not to be destroying, but to gain a fresh perspective from a frank re-evaluation of his own work and the work of his confreres. Much progress needs to be made and the critic can indicate the directions in which the investigators and the teachers should extend their best efforts. At the present time, I think that a fair critic would say that those interested should strive for a better understanding of the physiology of the nose, the relationship of the nose to the function of the lungs, the heart, and the abdominal organs; and secondly, that the knowledge which is available be offered to all qualified men who practice ear, nose and throat and those who are plastic surgeons doing rhinopolasty, and to provide for them a forum for the free exchange of ideas. Third, the critic, I believe should emphasize the fact that the rhinologist in rhinoplasty has a sphere very definitely his own. This sphere of interest originates in his fundamental training in the basic sciences of otolaryngology and enlarges from his studies and experiences in septum surgery, sinus surgery, nose and ear disease and the consideration of the whole problem of nasal obstruction. His eventual achievement must be a broad conception of nasal physiology upon which to predicate his further medical and surgical practices. The rhinoplastic surgeon on the other hand has his origin in general surgery and his training includes orthopedic surgery, oral surgery, and breast surgery and he comes to rhinoplastic procedures through his work in total and partial reconstructive and replacement surgery. Thus these two specialists meet in a similar field but from two widely different points of view.

The rhinologist, as was indicated above will reach his fullest development through the revelations of nasal physiology while the rhinoplastic surgeon will find his life's work in the broad field of general plastic surgery. Each can help the other realize his complete destiny. Each alone has found it difficult.

As I look back over 30 years of rhinologic surgery and compare it with the present trends I feel that the rhinologist has taken on a fifth role—that of creating a new and vigorous member of the otorhinolaryngologic family.

FURTHER OBSERVATIONS ON NASAL CILIA.*

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Throughout the ages the source of mucus which bathes the respiratory and gastro-intestinal tracts has been a point of interest to anatomists.

Without the aid of the microscope it was only natural for Hippocrates, Galen and their followers for over a thousand years, to think that nasal secretion was formed in the brain and filtered through the sieve-like cribriform plate to bathe the lining of the nose.

The moisture in the lungs and the glistening appearance of the mucous membrane of the trachea was also noted by them and thought in some way to be due to the liquids taken by mouth.[1]

It was not until 1660 that Schneider described the nasal mucosa as the source of mucus. He stated that "Mucus may be squeezed out of the membrane of these regions even in the dead subject."[2] Unfortunately he was unaware that the serous and goblet cells and racemose glands of the membrane were responsible for this secretion.

At about the same time in the Netherlands, an obscure layman by the name of Leeuwenhoek, who was the janitor in the town hall in Delft, Holland, began cutting lenses. This man was so obsessed with the idea of grinding better lenses that over 300 convex magnifying glasses were found in his house after he died. Leeuwenhoek studied any minute objects that he could lay his hands upon, such as grains of sand, cloth, butterfly wings, insects, seeds, pieces of hair, etc., and was amazed to find that blades of grass appeared

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like telegraph poles under his lenses. His efforts were crowned with success because he finally developed a compound microscope about the year 1675. A compatriot of his, Anton de Heide, described cilia for the first time in 1683, and 12 years later (1695) Leeuwenhoek, the janitor, noted ciliary movement. Until that time nothing was known of histology, so there is little wonder that Schneider did not realize the importance of the serous and goblet cells in the formation of mucus.

Nearly 30 years passed (1724) before Santorini discovered the glands in the nasal mucosa under the microscope, which was in general use by this time. In describing the mucous-secreting glands Santorini stated, "If we wish to demonstrate them most clearly we arrange the membrane, previously cleansed and somewhat macerated under the crystal, and the light being conveniently and adequately arranged for it, we may recognize their number, their size and their color. Their size varies indeed, some being a little ovoid, and these are about equal to a grain of mustard."[3] (This could almost be a quotation from a modern text book on histology.)

For over a hundred years there was no further mention of cilia or their action. In 1830 Sharpey[4] made some careful observations on ciliary motion in the sinuses and trachea of dogs and rabbits which laid the foundation for our present knowledge. Since 1890 a number of articles have appeared in the literature showing a greater interest in this subject. The importance of a healthy ciliated mucosa with its overlying blanket of mucus in warding off infections has gradually become universally recognized.

In a previous article[5] a few comparisons were made between ciliary activity in certain lower forms of animal life and that found in the human nose. It is freely admitted that the stimulating mechanism which produces this highly coordinated movement is unknown at the present time. This applies to human nasal cilia as well as to cilia on the body surfaces of sea-water mollusks. Although it is unwise to carry the comparison too far, as has frequently been men-

tioned, the fact remains that ciliary beat can be well maintained for hours in both isolated animal and human tissue if kept submerged in Ringer's solution or even normal saline.

This, of course, points to an inherent property of ciliated cell protoplasm which is independent of nerve control.

Many theories have been advanced concerning the activating agent of cilia but up to the present time none have been universally accepted.

Even under the phase contrast microscope with oil-immersion lens which gives clearer definition, the structure of ciliated cells has the same appearance when the cilia are beating as when they are still. A few words about some of the advantages of the phase microscope might be in order. Under the usual standard type instrument fresh, unstained tissue often does not show enough differentiation to be appreciated. For instance, ciliated cells of nasal mucosa refract light poorly, so there is little contrast between cell outline, granular cytoplasm, nuclei and cilia. For this reason various stains have been used histologically in order to make cells and their contents stand out.

In recent years the phase contrast microscope has been developed. It is similar to the average microscope in appearance but differs in the amount and direction of light transmitted through the instrument. This is accomplished by means of discs or plates inserted in the objectives and the aperture stops in front of the condenser. As an example, under the direct beam of light in the standard microscope, a group of red blood cells appear like homogeneous masses resembling pancakes, while under the phase microscope with diffraction plates in place, the light blocked off in the centre and coming in from the sides gives a halo appearance to the edge of the erythrocytes—in other words greater contrast. Even with this instrument the granular protoplasm of human nasal columnar cells seems identical when the cilia are moving actively forward and backward as when they are paralyzed with cocain. Our lack of knowledge concerning ciliary movement is aptly stated by Gray,—"When

we remember that really nothing is known of the machinery whereby a muscle develops a tension when it is stimulated, it is not surprising that the ciliary mechanism also remains obscure. In one respect, however, ciliated cells invite more sweeping speculation than is usually expended on muscular units for, in contradistinction to the latter, the morphological structure of the cells is extraordinarily constant no matter what is the organ or the animal in which they are found."[6]

An important difference between muscular action and ciliary movement lies in the fact that muscular action takes place either in water or air whereas cilia move only in water. Many microscopic aquatic animals, such as Paramecia, of necessity need a water medium for the cilia on their surface to function and move them about. During the active movement of groups of cilia, a Paramecium moves in the opposite direction to the effective stroke, while the water close to the animal tends to move in the direction of the beat.

It is similar to a swimmer doing the crawl and pushing the water behind him as he moves ahead. If one of these marine animals is held stationary and the cilia are moving actively, they produce water currents over the body surface from head to tail. In other words it is like the paddle wheel on a river boat. When the steamer is in the open stream the paddle wheel moves it forward but if it is tied up to a dock with the engines running, the water is propelled along its sides.

Clinically, in the same way human nasal mucosa requires an adequate supply of mucus and intracellular fluid in order for the cilia to function properly. Dry spots in the nose such as occur in atrophic rhinitis mean that the cilia in these areas are either sluggish or have been destroyed altogether.

The mechanism producing ciliary movement appears to be the same in all animals whether on the outer surface of minute sea urchins or throughout the respiratory tract of man.

A few words concerning the structure of a typical cilium might be in order. Over 40 years ago a cilium was described as a protoplasmic extension of a surface cell which was

homogeneous in character. Even when seen by transmitted light or under dark field illumination there is no apparent differentiation.

Under polarized light there is clearer definition due to refraction along the edges of each cilium.

This is in all probability caused by light diffraction at the sides and not due to the cell structure. A similar appearance is noted under the phase contrast microscope.

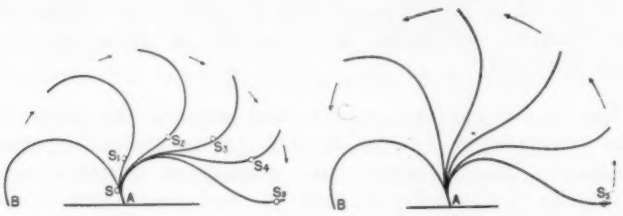
Although the actual exciting influence of ciliary movement is still a mystery it is interesting to consider an individual cilium during the effective stroke.

It should be remembered that a cilium is so minute that any thought concerning its substance is more theoretical than practical.

One of the earliest concepts and perhaps the simplest is that suggested by Schäfer[7] in 1891. In his original hypothesis he stated: "If we suppose that a cilium is a hollow curved extension of the cell, occupied by hyaloplasm, and invested by a delicate elastic membrane, then it must follow that if there be a rhythmic flowing of hyaloplasm from the body of the cell, into and out of the cilium an alternate extension and flexion of that process would thereby be brought about. . . . The same result might be got supposing the cilium to be a straight and not a curved extension of the cell, if the enveloping membrane were thicker (or otherwise less extensible) along one side than along the other."

A number of investigators refused to agree to this theory because they were unable to detect any liquid axis in any cilia they examined. One of the most strenuous objectors was Putter.[8] Justifying his original concept Schäfer maintained that "in all cilia or organs resembling cilia in which, on account of their size, it has been hitherto possible to observe any structure at all, they reveal themselves as tubular extensions of the cell substance enclosed by an elastic cuticula or pedicle."

Schäfer supported his contention by a number of models the simplest of which was a hollow rubber tube curved when in the position of rest. When water was forced in the tube it straightened out and when pressure was released the tube flew back to the position of rest at a speed independent of the rate at which it was straightened. Again if the pressure were applied slowly the tube was straightened again, but in this case the whole of the tube was stretched instead of only one side. This theory is interesting but it has its limitations. According to Gray,[9]—"The real value of Schäfer's hypothesis appears, however, to rest on two important facts: (1) the elasticity of the cilium plays an essential role, (2) relaxation as well as contraction is an active process."



Model of ciliary movement. *AB* is a strip of steel fixed at *A*. The strip is subjected to a bending force by the movable stop *S*; the bending force is applied at the base but gradually extends to the tip as the stop moves from *S* to S_5. On removing the stop the steel flies forward as in (*b*). Note that this model is of strictly limited value.

Figure 1

The other theory which has received the support of many workers is the one propounded by Heidenhain[10] in 1911. He considered a cilium as having a central elastic core with a contractile surface. According to Heidenhain "the effective stroke is produced by the active contraction of the sheath on that side of the cilium facing the direction of the effective stroke, and that the cilium regains its resting position, when relaxation takes place, by virtue of the recoil of the elastic core."

From the foregoing it is at once apparent that the views of Schäfer and Heidenhain are diametrically opposed: accord-

ing to Schäfer the mechanism which activates the cilium is derived from the cell itself and the work accomplished is due to the elasticity of the cilium; on the other hand, Heidenhain attributed the cilium as the potential source of energy when stimulated by the cell or from outside the cell. In other words, Schäfer considered the cilium as a passive sheath controlled by the cell while Heidenhain regarded the cilium as an active agent capable of autonomous contraction as a unit or part of a unit.

The main objection to Schäfer's hypothesis lies in the fact that he assumes that a cilium is a hollow tube which extends from the base to the tip. According to Erhard,[11] Prenant[12] and Saguchi,[13] both cilia and flagella are optically homogeneous when viewed by transmitted light or under dark field illumination. Even under polarized light there appears to be no differentiation in the internal structure of cilia or flagella.

A word or two about metachronal rhythm might be in order. This is a term that is little known by clinicians although frequently used by those interested in ciliary movement in animals.

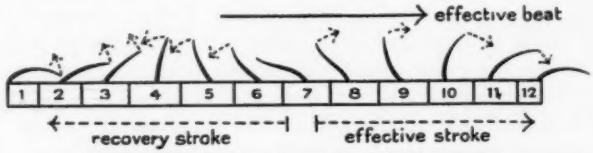


Diagram to illustrate metachronal rhythm. Cilia 1 and 12 are at the end of the effective stroke; 2–7 indicate successive stages during the recovery stroke; 8–11 indicate stages during the effective stroke. All the cilia 1–12 are beating in sequence.

Diagram illustrating the optical appearance given by a profile view of cilia beating in metachronal rhythm. (After Verworn.)

Figure 2.

Fresh human nasal mucosa examined under a high power microscope shows cilia beating at the same rate but not

beating in the same phase. The movement of groups of cilia is co-ordinated in a remarkable manner.

Each cilium moves without interfering with the one ahead of it and is slightly in advance of the one behind it. In this way regular waves are set up and this motion is known as metachronal rhythm. This type of sequence is found in almost all ciliated epithelium and has often been likened to a field of grain when the wind passes over it.

The crests of the waves show cilia at the high point of their effective stroke while the troughs are cilia at or near the beginning of the recovery stroke. This synchronization of movement suggests that there is some connection between the ciliary rootlets of one cell with that of its neighbor.

Although our knowledge concerning the mechanism of ciliary movement is very limited, a few definite facts stand out. Unfortunately, great gaps remain to complete the picture. Ciliated cells are such minute structures that careful microdissection is extremely difficult. We know, however, that cilia attached to a cell can beat independently of the cell as a unit.

Just before the turn of this century, Peter[14] observed that a cell could be damaged or crushed into small pieces without destroying the ciliary beat. He found that ciliary movement continued providing the cilia were still attached to a fragment of cytoplasm. About the same time Engelmann[15] noted that the tails of spermatozoa showed active movement even after the nuclei had been destroyed. In view of these experiments it would appear that the active agent producing ciliary movement lies in the distal portion of the cell. Every animal cell consists of three principal parts, the cytoplasm, the nucleus and the centrosome. Of the three chief constituents of the cell, the cytoplasm usually forms the greatest mass, the nucleus is next in size while the centrosome is exceedingly small. In animal cells including humans, the cytoplasm which fills the cells is a granular semi-solid fluid which has the consistency of soft jelly. The second chief component of the cell, and the one which perhaps is more important than the

cytoplasm—is the nucleus. It has to do with cell division. The third and by far the smallest of the three chiefs organs of the cell is the centrosome or "central body". Although very small it plays an essential role especially during cell division. It might be considered to be the "spark plug" of the cell. In general the centrosome almost always lies in the cytoplasm frequently close to the nucleus; however, in ciliated cells the centrosome apparatus appears to be multiple, taking the form of the so-called basal granules or corpuscles. These structures, in which the cilia of certain epithelial cells are rooted, are thought to be derivatives of the centrosome. It must be assumed that during the transformation of the indifferent cell into a ciliated cell the centrosome became subdivided into a large number of single centrosome-like structures, the basal corpuscles. On a slide they appear as an irregular line of poorly delineated dark bodies which stain deeply but show no structural detail even under the phase microscope with oil immersion lens. Although this idea has not been definitely proven it seems probable. The fact remains nevertheless that in cells showing motility the centrosome apparatus appears as the kinetic organ of the cell. This was also demonstrated by Peter.[14] On stripping a layer of cilia from the distal protoplasm of a group of cells, ciliary movement ceased. As the basal granules are at or just below the surface they would appear to be the source of stimulation. In other words once the cilia are isolated from the basal granules they stop beating.

Excised strip of lateral epithelium of *Mytilus* (diagrammatic). Note that the cilia are active as long as they are in organic communication with the cells.

Figure 3.

A similar condition can be seen on the gills of Mytilus. Mytilus is a member of the bivalved mollusks. It is a species of sea mussel and it belongs to the same order as the edible

oysters, scallops and fresh-water clams. This group shows one of the most intricately developed ciliary mechanisms. By means of cilia, feeding currents are set up, suspended particles filtered out and collected into specific ciliary tracts, and conveyed to the oral opening of the digestive tract. When a strip of cilia is elevated from the surface cells just above the basal granules the isolated cilia show a lack of motility.

These views, however, have not been entirely accepted. In 1910 Erhard[11] found that in some ciliated epithelium the basal granules could be destroyed by heat without influencing the ciliary beat.

A simple method of observing ciliary activity is to slow down the motion of the cilia by adding a drop or two of 5 per cent cocaine to Ringer's solution or by increasing the viscosity of the medium by means of a little gum arabic or acacia. Movement can also be reduced by lowering the temperature or by increasing the hydrogen ion concentration within the cells. By means of an ethyl chloride spray the slide can be cooled down and the addition of a weak solution of hydrochloric acid to the medium will lower the pH in ciliated cells.

A clinical application of this last point might be mentioned. Our knowledge concerning the common cold is unfortunately so limited that treatment consists largely in relieving the symptoms as they arise. In most cases a filterable virus which has an affinity for lymphoid tissue seems to initiate the infection. It paves the way for the secondary invaders or pus formers and these produce the disagreeable symptoms and complications which are so common. During the first three or four days of an acute rhinitis or sinusitis the nasal mucus is as a rule strongly alkaline with perhaps a pH of 10 or 12. Pathogenic organisms seem to thrive and multiply when the secretion is alkaline. Any method at our disposal which will reduce the high pH of the nasal secretion to the normal slightly acid level, just under 7, seems indicated.

Bed rest, warmth and sleep all help to promote an acid trend in nasal pH. Ephedrine in saline ½ or 1 per cent

with a pH of 5 or 6 also helps to ventilate the nose and tends to neutralize the high alkalinity. If palpitation or other side reactions occur it is sometimes better to prescribe the ephedrine by mouth (Ephed. Sulf. gr. ¼ or ⅜) two or three times a day. Weak hydrochloric acid 10 minims by mouth or glutamic acid hydrochloride capsules, 5 grains, three times a day after meals also aid in restoring the normal acidity to the body fluids. This of course is assuming that a slightly acid reaction is desirable. On the other hand hot drinks of lemonade or orangeade have been prescribed for years in the treatment of the common cold. Although the fruit itself is acid the solution is soon changed by the body metabolism to an alkaline reaction. As an example citric acid is frequently given to make the urine alkaline.

Many acute infections can be aborted by these simple measures and if steam inhalations and increased fluid intake are added, the treatment is more effective. The addition of steam vapor supplies the necessary moisture to the respiratory tract. This is just as important to ciliary activity in humans as sea water is to the cilia of Mytilus edulis. If the nasal mucosa becomes parched and dry, movement of the cilia is labored and sluggish.

Another similarity between the cilia of Mytilus and those found in the human nose is worthy of mention. A scrutiny of the literature failed to reveal any reference to it.

Cilia seem to develop more in certain fields than in others, depending upon the amount of work required of them. For instance, in some protected areas such as the latero-frontal gills of Mytilus the cilia are relatively short. Where more strenuous work is required the frontal cilia on the gills of Mytilus are longer and stronger.

In the same way the sinuses in man are not exposed to the outside air except very slightly on expiration. Here the cilia although well developed and packed tightly together are all about the same length but are relatively short. Where the load is heavy such as in the meatuses, due to the collec-

tion of mucus from the whole nasal mucosa including the sinuses, the cilia appear to be more powerful, longer and regularly spaced.

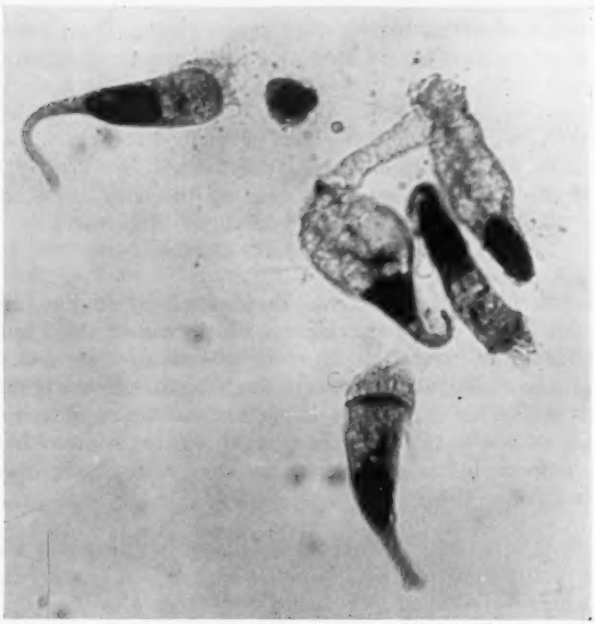


Fig. 4. Human nasal ciliated cells from the middle meatus. Note the relative length of the cilia in comparison to the size of the cell. The basal granules are also well shown in the lower perpendicular cell. Just above it is a goblet cell (magnified 1,000 times).

An attempt has been made in this article to try to present some of the theories on cilia and ciliary movement. A few of the time honored remedies in treating the common cold have been mentioned and their use in treatment appraised.

SUMMARY.

In summarizing our findings we must admit that there are many gaps in our understanding of ciliary activity; however,

little by little, our knowledge has increased so that our appreciation of cilia and their movement is much greater today than it was thirty years ago.

Gradually rhinologists are becoming aware of the tremendous amount of work accomplished by nasal cilia and the importance of their function. This marvellous mechanism of ciliary movement goes on day and night throughout the lifetime of every human being. Groups of cilia move while others rest and in this way cleanse the whole respiratory tract. Our very existence depends on the proper functioning of these microscopic structures so that it is vital for us to keep them in as healthy a state as possible. Our knowledge which has been materially aided by a study of certain marine animals, has given us a greater appreciation of nasal cilia and as a result a more conservative approach in treating rhinological conditions.

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ENDOSCOPY IN INFANTS AND YOUNG CHILDREN.*

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A great deal has been written about the many phases of endoscopy in infants and young children. The indications, dangers, and methods have been expounded and summarized, but I feel it is desirable to present again, briefly, the problems involved so that we may be ever mindful of the importance of endoscopic examination in the young. Those who do a great deal of endoscopy are probably more aware of the problems than those in our specialty who have the opportunity to do only occasional cases. If only for the latter, a review is warranted.

We are too inclined to fear this performance in infants and young children. The child is too sick, or the child is so young, or the infant is so small; these are thoughts that occur to us when we suggest an endosocopic examination; but with proper training and skill, with the use of adequate instruments, and with the expenditure of a minimal amount of time, there is little danger to the patient. Certainly following instrumentation, there is the possibility of subglottic edema that may necessitate a tracheotomy, but this can be largely overcome by gentle manipulation, using a tube small enough so that no trauma will result and keeping the instrument in place as short a time as possible. We ought to be able to accomplish an examination in about two minutes, and most foreign bodies should be extracted in less than five minutes.

There is still another factor in the avoidance of edema of the larynx, i.e. anesthesia. Certainly infants should be examined without anesthesia since it is possible to "mummy"

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them and keep them relatively quiet during the procedure; but for older children, perhaps above three or four years of age, I believe a general anesthesia is desirable. This reduces the possibility of trauma. We have been using ether anesthesia or a combination of rectal avertin and ether anesthesia or avertin alone. These are very satisfactory in experienced hands. Helrich, Daly, and Rovenstine are very enthusiastic about the use of rectal surital. We have no experience with this drug as yet. Intravenous pentothal in children is extremely dangerous, and we have seen some frightening experiences due to laryngo- and broncho-spasm. The desirability for general anesthesia holds for laryngoscopy and esophagoscopy as well as bronchoscopy. Certainly this must be dispensed with in a child that has acute laryngeal obstruction or an acute bronchial infection, but whenever possible we believe a general anesthesia is preferable; besides, the psychological trauma incurred is tremendous and should be considered in any operative procedure upon children.

ENDOSCOPY IN THE NEWBORN.

The most frequent difficulty encountered in the newborn is bronchial obstruction due to aspiration of amniotic fluid, meconium, blood, mucus, or vernix caseosa. In most instances it is important that the obstruction be relieved within a few minutes of birth. It is impossible to await the arrival of an endoscopist to perform this procedure; therefore, obstetricians must resort to the use of aspiration by catheter or exposing the larynx with a Flagg-type of laryngoscope and placing a catheter or an aspirating tube into the lung. Fortunately, obstetricians have become very adept at the procedure and it is successful most of the time. Recently, however, we saw a case that was aspirated with such vigor that an edema of the epiglottis resulted, necessitating a tracheotomy two days after birth.

If respiratory difficulty with cyanosis persists or recurs with the removal of oxygen, or if the respiratory embarrassment is intermittent, the role of the endoscopist is more

evident. It becomes necessary to examine the larynx and bronchial tree very thoroughly to determine the reason for the continued respiratory failure.

The importance of cooperation between the obstetrician, the pediatrician, the roentgenologist, and the endoscopist cannot be over-emphasized. Wherever possible, an X-ray should be taken in an effort to demonstrate the cause of the dyspnea.

The examination is done with relative ease, first noting the condition of the larynx, and then passing a bronchoscope suitable in size for the newborn. With average skill and training one can perform the procedure quickly and with no significant trauma.

In the larynx, after aspiration, we would note whether there is a congenital deformity such as exaggerated infantile larynx, yielding aryepiglottic folds, flaccid or elongated epiglottis, congenital web, paralysis of one or both vocal cords due to local or cerebral injury, congenital cyst, or a benign tumor. In addition to difficulty in breathing some of the above conditions produce a laryngeal stridor. Active therapy may not be required immediately and sometimes not at all. The stridor stabilizes and, especially in the infantile larynx, disappears in time, but only by means of direct laryngoscopy will the exact diagnosis be made.

Laryngeal obstruction is sometimes due to trauma as in the instance I have already mentioned, and at other times due to infection. We have seen a case of thrush of the mouth and pharynx that extended to the larynx and necessitated a tracheotomy when the child was less than two weeks old.

If the larynx is found to be normal, the bronchoscope is passed. Thorough aspiration is accomplished to remove as much of the material in the bronchi as possible. Oxygen is administered at the same time. The expansion of the lung may be incomplete in the early hours of birth sometimes due to over-medication of the mother or to the anesthesia during labor. Especially in difficult labor or birth injury

there may be a lack of forceful respiratory efforts that are necessary to separate the sticky alveoli. In any case bronchoscopy with aspiration and oxygen administration will frequently decrease the extent of the atelectasis enough to prolong life until the infant is able to thrive on its own.

The atelectasis may be unilateral or bilateral, and here we wish to emphasize the importance of the X-ray. Frequently respiratory failure may be considered atelectasis secondary to aspirated material when as a matter of fact an X-ray will show that there is a congenital defect in the diaphragm with abdominal contents in the thoracic cage. Perhaps the X-ray will demonstrate the presence of a pneumothorax secondary to a partial bronchial obstruction or a congenital cyst of the lung. On rare occasions we may encounter a case of agenesis of one lung or a portion of a lung. Then too, we must keep in mind the possibility of enlarged thymus. This diagnosis can be made only when other possibilities have been excluded by laryngoscopy and bronchoscopy.

Respiratory failure may also be caused by injury to the central nervous system at birth or compression of the neck by the umbilical cord. The history of the type of birth is most important.

Congenital atresia of the esophagus accompanied by a fistula into the trachea may become evident shortly after birth when swallowing mucus or food produces coughing and cyanosis. X-ray with lipiodol instillation into the esophagus, and endoscopy, help to make the diagnosis and localize the pathology.

LARYNGOSCOPY IN YOUNG CHILDREN.

In the young child we are occasionally able to examine the larynx by the use of the laryngeal mirror, but direct laryngoscopy becomes necessary in a large percentage of cases. As we have mentioned before we prefer direct examination under general anesthesia. Of course, in the presence of severe laryngeal obstruction the procedure must be per-

formed without anesthesia. Local anesthesia, especially cocaine, is dangerous, and it seldom gives the desired results. Cases must be considered individually under these circumstances.

Acute laryngitis and ordinary croup do not require examination, but when laryngeal obstruction with its stridor, labored respirations, retraction of the intercostal spaces and supraclavicular areas, cyanosis, etc., is present, direct laryngoscopy becomes necessary to determine the cause. This must be done mindful of the possibility of asphyxia that may call for an immediate tracheotomy. A bronchoscope should be held in readiness to insert into the trachea if complete obstruction occurs during this type of laryngoscopy. Frequently it is possible to pass the bronchoscope and proceed with the tracheotomy with the tube in place; however, in cases where asphyxia is apt to occur during the procedure, we sometimes do the tracheotomy first and examine the larynx afterwards. In acute laryngo-tracheo-bronchitis the likelihood of membrane formation and crusting in the trachea and bronchi is very great, and the endoscopist must be available at short notice to insert the bronchoscope through the tracheal opening and remove the obstructing material.

The possibility of a foreign body must always be kept in mind. Edema of the epiglottis or larynx due to trauma, corrosive substances, angioneurotic edema, or infection requires direct examination. We have recently seen a membranous laryngotracheitis due to a pyocyaneus infection which after tracheotomy caused a great deal of crusting in the trachea and bronchi. The condition was eventually overcome by the use of Polymyxin and Tryptar by aerosol as well as repeated aspiration through the tracheotomy opening.

Stridor or hoarseness that continues over a period of time requires laryngeal examination. In younger children we frequently find the abnormalities of the larynx mentioned before, i.e. elongated and flaccid epiglottis, exaggerated

infantile larynx, collapsing aryepiglottic folds. Stridor in these cases disappears as the child grows older—from two to four years of age.

Congenital abnormalities such as web or cyst may be picked up during early childhood. Recurrent nerve paralysis may be encountered. The latter is most frequently caused by birth injury but may be due to pressure on a nerve along its course.

Benign tumors such as a papilloma, a hemangioma, a polyp, or a fibroma may be seen. These are readily visualized and removed by direct laryngoscopy. Here we wish to stress the importance of infection in the sinuses and tonsils, and changes in the upper respiratory tract due to allergy, and also the improper use of the voice, in producing inflammatory reactions in the cords. The findings may vary from a diffuse redness and edema of the vocal cords to hypertrophic changes, and on to inflammatory nodules, i.e. polyps or fibromas.

BRONCHOSCOPY IN THE YOUNG CHILD.

Bronchoscopy in children is indicated wherever there is uncertainty as to the diagnosis of a lung condition. Persistent unexplained cough, localized wheeze, hemoptysis, purulent expectoration are frequently explained by bronchoscopy. On the other hand, when a diagnosis of an abscess or bronchiectasis has already been made, a bronchoscopic examination is required in an effort to determine the cause and to promote drainage. Any suspicion of a foreign body calls for immediate examination and removal. X-ray findings of bronchial obstruction, atelectasis, emphysema, or pneumothorax must be examined, for in only this way are we able to ascertain the pathology. The suspicion of endobronchial tuberculosis or enlargement of peribronchial lymph nodes producing bronchial symptoms may be confirmed by direct inspection. Compression of the trachea or bronchus by mediastinal or pulmonary tumor, a lymphoma or cyst, enlarged thymus or enlarged lymph nodes, may be noted on bronchoscopy.

Noisy respirations should be investigated. If the trouble is not in the larynx when doing a direct laryngoscopy, a bronchoscopy is indicated and will reveal the difficulty.

Asthma and its accompanying thick secretions may be helped by aspiration, and at the same time one is able to exclude causes of noisy respiration other than asthma.

In bulbar poliomyelitis with respiratory paralysis exudate accumulates in the lung and must be aspirated. If repeated aspiration is necessary, a tracheotomy is recommended to facilitate the cleansing of the bronchial tree.

Persistent pneumonitis or recurring pneumonitis may be due to a foreign body or some other form of bronchial obstruction.

By bronchoscopy we are also able to take specimens of exudate for culture and examination. In this way we may confirm a diagnosis of tuberculosis by direct smear.

At the time the bronchoscopy is performed lipiodol may be instilled and X-rays taken when a bronchogram is indicated. This is especially important in the diagnosis and localization of bronchiectasis, and is of great significance to the thoracic surgeon.

Certainly bronchoscopy is essential to the chest surgeon both from the standpoint of diagnosis and of localization of the pathology. Pre-operative and post-operative bronchoscopic aspiration is of great import. Post-operative atelectasis calls for immediate aspiration.

ESOPHAGOSCOPY IN THE YOUNG CHILD.

Esophagoscopy in the young child is most frequently employed for the removal of a foreign body. Whenever a foreign body is suspected, even though not demonstrable by X-ray, an examination should be performed.

I repeat that wherever possible I prefer to do the procedure under general anesthesia. It is done with greater ease and with much less danger to the child. To extract

a foreign body while the patient is twisting and turning makes the possibility of injury to the esophageal wall far more likely.

Strictures of the esophagus due to corrosive substances must be dilated by esophagoscopy. The question of early esophagoscopy in cases of esophageal burns is a debatable one. We prefer to do the examination as soon as the acute symptoms have subsided and dilatation is carried out following this, according to the condition observed.

Congenital abnormalities such as partial stricture and webs are encountered by the endoscopist. These may be seen soon after birth or may not be recognized until early childhood.

Cardiovascular anomalies may produce tracheal, bronchial, and esophageal obstruction. The diagnosis is frequently made or confirmed by direct examination. Certainly the picture of the extent of the existing pathology is greatly aided by the endoscopist if only to exclude other possibilities that may cause the obstruction.

CONCLUSION.

An attempt is made to review the subject of endoscopy in infants and children. The scope of the paper is such that the indications, methods of examination, and diseases are mentioned only briefly. The intent is simply to indicate the importance of endoscopy so as to encourage its more frequent use and to allay the fears of those who hesitate to recommend or perform the procedure.

114 East 72 Street.

ELECTROLYTE STUDIES IN MENIERE'S DISEASE.*†

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The pathogenesis of Meniere's disease is unknown. Despite a great deal of astute clinical observation and experimentation the unique symptomatic manifestations of this disease cannot be clearly explained. In recent years one major feature of the pathology has been clearly defined and places the primary site of the disease in the inner ear. The dilatation of the membranous labyrinth, particularly of the cochlear duct and saccule, has been widely reported and the term "labyrinth hydrops" is now often used synonymously with Meniere's disease; however, more structures might be affected, but the exact state of the hair cells, stria vascularis, endolymphatic sac and other finer details here cannot be assessed satisfactorily because of post-mortem degeneration. Dix and Hallpike[1] (see Fig. 1) believe that hair cell changes can be demonstrated and that widespread hair cell damage is present in Meniere's disease and probably explains the marked recruitment that is associated with the deafness in this disease.

Because of our meagre understanding of this disease, treatment has been empirical and greatly varied. The fluctuating nature of the symptoms with periods of remission, and the chronicity, makes evaluation of treatment difficult. While the audiogram provides a good index of change in the cochlear part of the disease, caloric tests are a less useful measure of vestibular dysfunction particularly

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in the early stages; furthermore, the vestibular and cochlear symptons may not always correspond. An ear that shows a marked hearing loss may nevertheless still be producing severe vestibular attacks.

For many years major attention has been directed to the salt and water metabolism in this disease and the treatment has been based on real and speculative pathological findings of such metabolism. The first careful study of this problem cn a group of hospitalized patients by Mygind and Dederding[2] some twenty years ago seemed to indicate a definite relationship between water retention and aggravation of cochlear and vestibular symptoms. A few years later Furstenburg, and Lathrop[3] reported "that the basic disturbance in patients with Meniere's disease was sodium retention and that apparently the local tissues involved in Meniere's disease have either an increased avidity for sodium or an unusual sensitivity to it." They claimed to have regularly produced attacks of vertigo when administering a single large dose (15 gr.) of sodium, as bicarbonate or chloride, in each of 14 patients, whether dehydrated or well hydrated and before the weight gain due to secondary water storage occurred. Later Talbott[4] reported that the blood sodium in patients with Meniere's disease was normal during and between attacks and hence concluded that this was not a disease aggravated by a disturbance of sodium metabolism. He found the blood potassium high in some patients during an attack of vertigo.

As the result of a few isolated studies of this type, a generally accepted method of medical treatment evolved that was concerned with restriction of salt intake, usually to 3 gm. daily. There is also wide use of ammonium chloride to aid in elimination of sodium and of potassium chloride to attempt to raise the blood potassium. While in general this has been the basis of medical management and is widely practiced, the otologist's understanding of the actual changes in body electrolytes that occur under these conditions has not been clear. Since these few pioneering clinical investigations, no further carefully conducted clinical studies on

Meniere's patients as to the effect of controlled changes in body electrolytes have been reported. On the other hand, in these past fifteen years, improved and more accurate methods of changing and measuring electrolytes in the body have been developed and are widely used by the internist especially in the diagnosis and management of metabolic diseases.

At the same time newer experimental observations on the inner ear stress the importance of electrolytes such as sodium and potassium in the normal function of the labyrinthine end organs. Thus recently the investigations of Smith[5] and her associates on the relative concentrations of sodium and potassium in the perilymph and endolymph indicate that unlike ordinary extracellular fluid and closely resembling intracellular fluid there is over 20 times the concentration of potassium in the endolymph (140 mil. eq/1.) as in the perilymph (6 mil. eq/1.). On the other hand the concentration of sodium in the endolymph (15 mil. eq/1.) is only a tenth as high as that in the perilymph (150 mil. eq/1.). Sodium and potassium concentrations in the cerebrospinal fluid closely resemble that in the perilymphatic space (see Fig. 1). In the intracellular fluids of most cells, there are about 7 milli equivalents of sodium to about 100 milli equivalents of potassium.

This electrolyte pattern in the labyrinthine fluids, unique among known extracellular fluids, probably has some vital significance in the function of the sense organ. The active or passive role of Reissner's membrane and of the stria vascularis, for example, must be considered in the maintenance of this sharp electrolyte gradient between the two fluids. Recent experimental work by Tasaki and Fernandez[6] confirms an earlier report that the perilymph can be completely replaced by Ringer's solution without altering the electrical responses of the cochlea to sound; however, they observed that these electrical responses can be markedly suppressed by the addition of a .25 per cent solution of K Cl to the Ringer's. The normal concentration of potassium in Ringer's solution is about one sixth of this amount.

The importance of a normal electrolyte concentration in the labyrinth fluids for the functional integrity of the many complicated labyrinth end organs is becoming more and more clear.

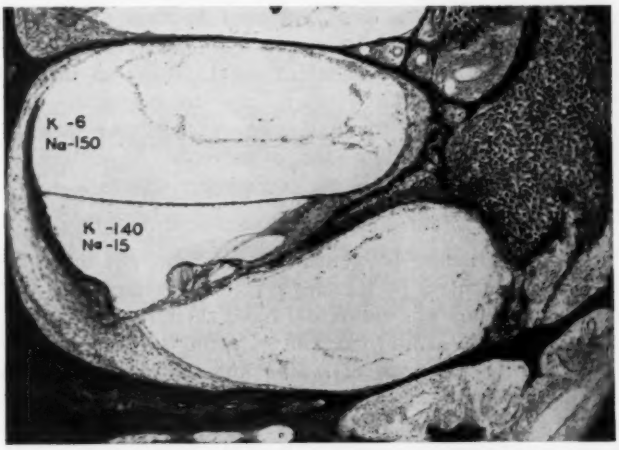

Fig. 1. Photomicrograph of the cat cochlea indicating the sodium and potassium concentrations in the endolymph (cochlear duct) and perilymph (scala vestibuli) reported by Smith and her associates.

The unique property of the endolymph electrolytes is probably related also to the recent findings of Bekesy.[7] He has shown that the endolymph is at a constant higher electrical potential than the perilymph, that Reissner's and the basilar membrane electrically insulate the endolymph-charge from the perilymph. He found that anoxia reduces the constant direct current potential in the endolymph. In contrast to the positive potential of the endolymph fluid, Bekesy reports that the cells of the organ of Corti and of the stria vascularis are at a negative potential relative to the perilymph. These recent experimental observations give us a glimpse of some of the electro-chemical factors that play a vital role in the normal function of the labyrinth end organs and some insight into the kind of disturbances that may change their function. The explanations for the fluc-

tuations in auditory function in Meniere's disease, for example, may somehow be hidden in these chemical and electrical properties of the labyrinth fluids. The marked discrepancy between the electrolyte concentrations of the endolymph and those of other extracellular fluids make it unlikely that there is free diffusion between the arterial blood supply and the endolymph. The close similarity of endolymph electrolyte concentrations to those of intracellular fluid should perhaps allow one to postulate similar variation in response to change in extracellular levels. If this theory is correct a sharp increase in extracellular sodium would have as one of its primary effects a dehydration of the endolymphatic space.

The normal human body has a remarkable set of mechanisms for the control of its electrolyte concentrations. While these mechanisms are many and have their origin in various organs and tissues, the final control point is the renal tubule where varying amounts of water and salt are reabsorbed, according to the need of the organism. A large salt intake, whether by mouth or by vein, results in temporary water retention and consequent dilution of the extracellular sodium and the maintenance of a constant sodium level. This extra salt is then promptly excreted with its proportionate amount of water. One can inject intravenously 10 to 15 grams of sodium in hypertonic solution with an insignificant change in serum sodium. On the other hand, prolonged low salt intake results in retention of nearly all sodium by the kidney. Consequently even severe restriction of sodium intake in the normal human will not quickly result in lowering of either total body or extracellular sodium levels.

Both water retention with hydremia and salt retention have been implicated in the pathogenesis of Meniere's disease. Neither of the groups who studied these situations inflicted very extreme conditions on their patients and neither measured the sodium concentrations which resulted. If these patients had normal homeostatic mechanisms it seemed doubtful that significant variations actually resulted from their procedures. These doubts were pointed up some

time ago by a patient who had adrenal insufficiency and active Meniere's disease. He had a number of acute Meniere's attacks at times when his serum sodium was measurably low and his serum potassium high. After his Addison's disease was brought under control and the electrolytes were kept at normal levels, the attacks of vertigo ceased entirely and there was some improvement in his hearing.

The role of electrolytes in Meniere's disease remains obscure and a careful re-examination of this problem seems indicated. Fluctuation in symptoms may be related to changes in electrolytes, and in patients with active disease under careful control, this relationship might be made more clear. The effect of any controlled change in the composition of the extracellular fluid as regards sodium and potassium on the clinical manifestation of this disease seems worthy of careful study, particularly in patients with an active stage of the disease.

Obviously such a study requires hospitalization and this is well justified, particularly in a number of these patients who had the disease in both ears and where the impairment of hearing was fluctuating but the hearing lesion was severe enough to be of even greater concern than the attacks of vertigo. Only very severe salt restriction is likely to cause a measurable change in the sodium concentration of extracellular fluids. Ordinarily, changes in the serum sodium are reflected rather promptly in the adjacent extracellular fluid spaces; therefore, a period of observation of two to four weeks in the hospital would probably be adequate to allow these changes to become manifest.

Our plan was to study the two extremes of sodium concentration in patients with active Meniere's disease in the sense that their hearing levels were still variable and they were still having periodic attacks of vertigo. The first group of patients, whose sodium levels were to be lowered were placed on diets containing at most 300 mgm. of sodium. Then they were given ammonium chloride, 4 to 8 grams per day and thiomerin 2 cc. every other day to prevent

normal sodium conservation by the kidney. Even with this artificial interference with normal kidney function it was quite difficult to reach and maintain a significantly lowered serum sodium level. The sodium excretion was markedly lowered and dehydration occurred in the body's attempt to maintain proper sodium levels, but by using sufficient mercurial for a long enough time, a moderate lowering of serum level was achieved. In a few cases where the rate of sodium loss was slow, intravenous aminophyllin was used with intravenous mercurial diuretic to effect an increase in the rate of sodium diuresis.

In the second group of patients extra dietary salt was provided in amounts up to 8 grams per day and the retention of this extra salt was brought about by treating the patients with varying amounts of desoxycorticosterone, a steroid which causes sodium retention probably by effecting more complete sodium reabsorption by the renal tubule. When water intake was not restricted the patients also retained water in an attempt again to maintain a normal sodium concentration but in all cases a significant increase in serum sodium concentration was produced. If the theories of Mygind and Furstenberg were correct, the clinical situation produced by these procedures should have been therapeutically effective in ameliorating the disease in the first group or should have aggravated the disease in the second.

Potassium deficiency has also been implicated by Talbott et al[4] as playing a role in the pathogenesis of this disease, and on the basis of his reports oral potassium is used by many as an additional therapeutic agent. We have not attempted seriously to distort potassium balance in these patients but one of the major effects of desoxycorticosterone "doca", in addition to causing salt retention is a marked potassium diuresis, so that patients given large amounts of doca should have changes in sodium, potassium and water metabolism.

Mygind and Dedering used a water test in an attempt to show a disturbance in the ability of Meniere's patients to dispose of extra water. The test consisted of the ingestion

of 1000 cc. of water and the measurement of weight and hematocrit changes after four hours. They stated that their patients with Meniere's disease retained more than a normal amount of water and their symptoms were aggravated during this procedure. In order to evaluate this conclusion, we have used in our patients the Kepler water test which was devised at the Mayo Clinic and widely used the country over as a test of adrenal function. This consists of giving orally 20 cc. of water per kilo to patients who have been without water for 15 hours and measuring their urinary output during these 15 hours and at hourly intervals for four consecutive hours after the test water is taken. A normal patient will excrete more urine in one test hour than during the 15 hour preparatory period. The test has been standardized on many hundreds of normal people in many clinics and has been shown to give an accurate measure of water diuresis. None of these patients showed a change in their auditory threshold curve or in vestibular symptoms during the Kepler water test which was performed prior to therapy.

We have used these procedures in a total of 15 patients causing in all cases significant drops in serum sodium and total body water or after the administration of doca, moderate increases in those constituents. A typical result of these measures is seen in Fig. 2. There was first noted a decrease in body weight as an attempt was made by the body to maintain a normal sodium level. As sodium conservation was prevented by the mercurial diuretic the serum level fell. Then as extra sodium was made available and sodium excretion was prevented by desoxycorticosterone "doca" there was marked retention of water, again in an attempt to maintain a normal sodium level. The degree of retention was such that a sharp rise in serum sodium level resulted. The degree of change in total extracellular sodium was really much greater than serum levels show since the acute weight changes are chiefly losses or gains of extracellular fluid. On the average a 50 kilo person has about 12 liters of extracellular fluid containing about 1700 milli equivalents or 39 gm. of salt. The approximate loss in this patient can be

calculated to be about 13 gm. or one third of her initial extracellular sodium. As she rapidly gained 6 kilos, the sodium retained with this water was about 25 gm. representing an increase of about 100 per cent in her actual total extracellular sodium.

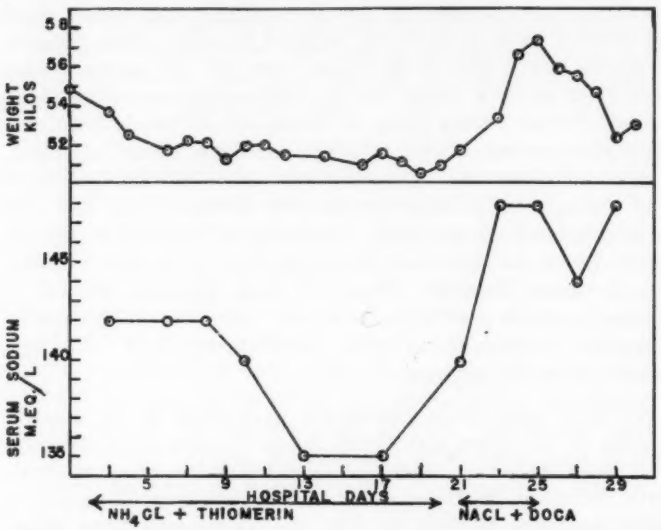

Fig. 2. Graph of weight change (upper curve) and serum sodium levels (lower curve) in a typical patient depleted of sodium and then given sodium chloride and desoxycorticosterone. See explanation in the text.

We have been able to study the effect of either an increase or a decrease in extracellular sodium and water on the clinical manifestations, particularly on cochlear function which was tested almost daily. To date we have data on 15 patients who have been under careful observation in the hospital for periods of from two to four weeks. Observation on the effect of a period of sodium depletion immediately followed by a period of sodium retention were also made in a number of cases. All of the patients presented signs of active labyrinth involvement as indicated by lability of both vestibular and cochlear function. Six of the patients

had bilateral active disease, a seventh had severe active disease in her remaining live ear; the cochlear function of the opposite ear having been destroyed earlier by mumps.

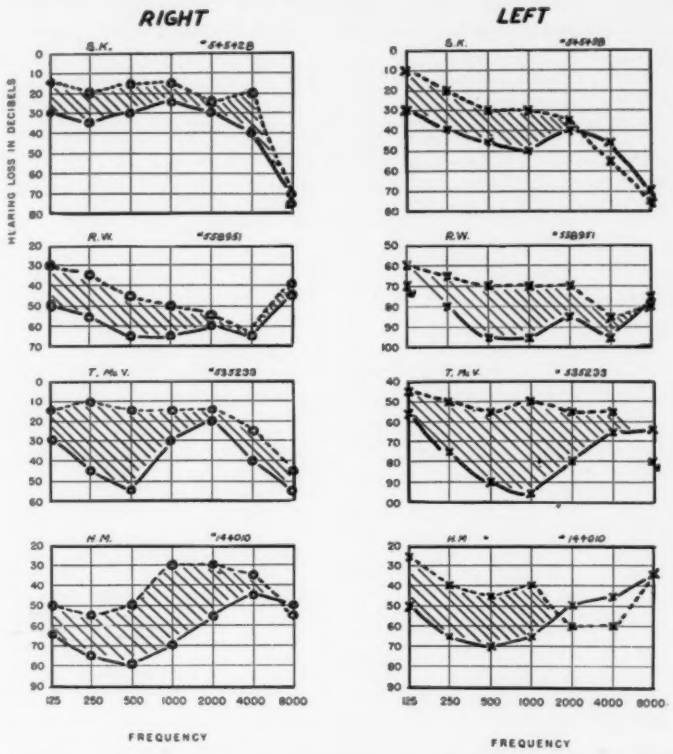

Fig. 3. Audiograms of four patients with active bilateral Meniere's disease in the group studied showing the degree of auditory threshold fluctuation during their hospital stay. Note the different optimum levels in the decibel scale.

Auditory function tests were carried out by Mr. Duncan Scott.

The lability of the disease was evident in all patients during their hospital stay, particularly as to cochlear function. The auditory threshold changed frequently and this fluctuating deafness was clearly evident in the patients with bilateral disease when talking with them. Tests indicated

that the intelligibility of speech was particularly poor ("strong recruitment") in these and other cases of Meniere's deafness. The degree of auditory lability is demonstrated in Fig. 3. Some of the patients while in the hospital with a measured depletion of their serum sodium had severe attacks of vertigo.

While the group whose sodium level was lowered showed fluctuations in hearing, the patients on desoxycorticosterone showed either no change or in some cases an improvement in their symptoms. A number of patients who were maintained at a low sodium level for some time without improvement in hearing and with attacks of vertigo showed an improvement in hearing and in auditory and vestibular symptoms when their electrolytes were allowed to return to normal.

While this report is a negative one, it does not imply that these induced changes in extracellular fluids are necessarily reflected in the composition of the labyrinth fluids. Neither does it refute the possibility that this disease is related to disturbances in the labyrinth fluids, particularly the endolymph. It merely indicates that present therapy directed at changing the electrolytes is probably ineffective and emphasizes the type of study that is necessary even to begin an evaluation of salt and water metabolism in this disease.

SUMMARY.

1. Fifteen patients with active Meniere's disease were observed in the hospital on programs that produced measured increases or decreases of serum sodium levels.

2. No consistent effects on cochlear or vestibular function were noted in patients with a measured low serum sodium as random fluctuations in hearing continued and some acute severe attacks of vertigo were observed.

3. No acute attacks of vertigo were seen in patients whose serum sodium had been sharply raised and no consistent deterioration of cochlear function was produced. A

number of these patients even had significant and sustained improvement of cochlear function during and following such treatment.

4. The Kepler water test failed to reveal impaired water diuresis in these patients and no changes in vestibular or cochlear function was noted during the course of the test.

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NEUROFIBROMA OF THE LARYNX.*† PRESENTATION OF FIVE CASES.

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Neurofibromas occur in many parts of the body; they may be solitary or a part of a generalized neurofibromatosis or von Recklinghausen's disease. Although involvement of the larynx by a neurofibroma is of infrequent occurrence, the disease may present definite diagnostic and therapeutic problems.

Colledge,[1] in 1930, reported a case in which laryngeal involvement occurred in the course of generalized neurofibromatosis. The patient was a 44-year-old woman who complained of progressive dyspnea and dysphagia. General examination disclosed numerous superficial tumors distributed along the courses of the cutaneous nerves. A patchy discoloration of the skin also was evident.

Laryngeal examination disclosed a well-circumscribed tumor mass occupying the right aryepiglottic fold. The tumor was encapsulated and was easily shelled out when exposed by an external approach.

Holinger and Cohen[2] in 1950, reported a case in which a pedunculated lesion of the pyriform fossa overhung the glottis and produced symptoms of obstruction. Removal, with the aid of the snare, was followed by postoperative bleeding. These authors suggested the advisability of electrocautery and ligation of the base as a means of avoiding this complication.

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The fact that involvement of the laryngeal tissue sufficient to give rise to serious symptoms is not common is indicated by the rarity of reports of such cases in the literature.

NEUROFIBROMAS OF LARYNX IN GENERALIZED NEUROFIBROMATOSIS.

Two cases in which laryngeal involvement by neurofibromatous tissue occurred in the course of generalized neurofibromatosis were found in the files of the Mayo Clinic.

REPORT OF CASES.

Case 1: A man, 41 years old, presented himself for examination with the complaint of hoarseness. He said that hoarseness had begun five years previously, at the time of surgical removal of a large mass involving the right side of the neck, from the mastoid region to the level of the clavicle. The diagnosis had been "neurofibroma."

General examination disclosed a number of shotty nodules palpable in the supraclavicular and posterior cervical regions. A roentgenogram of the thorax demonstrated a well-circumscribed mass in the right side of the thoracic cavity at the level of the third thoracic vertebral body with a broad base toward the mediastinum. These lesions were interpreted as being neurofibromas.

Laryngeal examination showed a diffuse fullness of both arytenoid muscles and aryepiglottic folds, extending downward into the postcricoid region. The right vocal cord was fixed. A tentative diagnosis of "neurofibromatous involvement of the larynx" was made. Examination of tissue removed under suspension laryngoscopy verified this diagnosis.

No attempt at removal of the lesions was indicated, in the absence of serious symptoms The possibility of doing more harm than good by performance of the radical surgical procedure that would have been necessary because of the diffuse nature and the extent of the lesions is quite apparent.

Case 2: A woman, 50 years old, presented herself for examination complaining of hoarseness of twenty years' duration, associated with a sense of irritation in the throat.

Since adolescence muliple subcutaneous nodules had developed. A diagnosis of "von Recklinghausen's disease" had been made.

Examination of the larynx disclosed a nonulcerated tumor involving the left arytenoid area, with apparent extension down the postcricoid region into the esophageal introitus. Under suspension laryngoscopy the tumor was removed with the cutting diathermy electrode. A pathologic diagnosis of "neurofibroma" was made.

A letter received two years after this operation stated that the patient was having no more trouble with her throat.

In these two cases the lesions were diffuse, with no definable border, and no evidence of encapsulation. In the case reported by Colledge[1] the lesion was well localized, discrete and encapsulated. Hence it appears that involvement of the larynx in cases of generalized neurofibromatosis may take one of two forms: either that of a well-localized, encapsulated lesion, or that of a diffuse, poorly demarcated lesion.

Neurofibromas also may be found involving the larynx in the absence of a generalized disease.

Since 1926, when Suchanek[3] reported his case of solitary neurofibroma of the larynx, a number of cases of involvement of the larynx by this type of tumor have been reported. Smith,[4] in 1944, presented three cases and reviewed the literature to find 12 additional ones. This review did not include cases reported by Eigler[5] in 1937, and by Baldenweck and associates[6] in 1942.

Walker,[7] in 1947, and Oliver, and his colleagues[8] in 1948, Fisher and Odess in 1949,[9] and Pearlman and his colleagues[10] in 1950 presented eight more cases to bring the total of 25 cases presented by 19 authors, prior to 1951.

The information obtained from study of the available literature indicates that the disease may occur at any age. The youngest patient was six years old and the oldest was 53 at the time of diagnosis.

Women appear to be afflicted slightly more frequently than men, in a ratio of about 3:2.

Symptoms are due to the physical presence of the tumor and so are determined by its size and location. They are hoarseness, dysphagia, a sensation of "something in the throat," dyspnea and stridor. The duration of these symptoms before diagnosis varies from a few months to several years.

The lesions, when made visible by mirror examination of the larynx, usually are rounded, smooth, well-circumscribed tumor masses which may be pedunculated. Jackson

and Coates,[11] however, described a lesion which presented many submucous nodules, and in the third case reported by Smith[4] the lesion appeared to be an irregular papillary growth. The lesions in the four cases of Pearlman and associates gave the appearance of laryngeal polyps. Hence, it is seen that there are three types of involvement: (1) the discrete encapsulated tumor, which is of most common occurrence; (2) the diffuse, poorly localized nodular involvement by neurofibromatous tissue; (3) the polypoid tumor of the vocal cord which, although well localized, is not encapsulated.

The overlying mucosa invariably is intact unless it has been traumatized by attempted excision of specimens for biopsy. The color may be red, yellow or gray.

The portion of the larynx most frequently involved is one or the other aryepiglottic fold or a ventricular band. Often the tumor overhangs the glottis, making it impossible to visualize the condition or the mobility of one or both vocal cords.

In five instances the involvement was limited to the free edge of one vocal cord.

The diagnosis of neurofibroma of the larynx is made by microscopic examination of tissue removal from the lesion. Because the overlying mucosa invariably is intact, and because the tumor frequently is well encapsulated, it is often difficult to obtain tissue for biopsy. The diagnosis, then, generally is made by examination of the tumor after it has been removed. The treatment is surgical removal. The well-encapsulated tumors are easily enucleated. The diffuse tumors and the polypoid tumors of the vocal cords are difficult to separate from the surrounding tissues.

The approach to the tumor may be transoral or by an external operation. Temporary tracheotomy may or may not be necessary.

Of interest is the observation that the vocal cord on the side of the encapsulated lesion may be completely or

partially paralyzed after the operation. The cause of this paralysis is not apparent when it is considered that the motor innervation of the larynx is by the recurrent laryngeal nerves, which do not come in proximity to these supraglottic lesions.

Vail[12] suggested that this type of paralysis is more likely to result if an external, rather than a transoral, approach is made for removal of the lesion.

SOLITARY NEUROFIBROMAS OF LARYNX.

Three cases in which solitary neurofibromas of the larynx had occurred were found in the files of the clinic.

Case 3: A woman, 49 years old, presented herself for examination complaining of hoarseness of twelve years' duration.[13]

A thickening at the midportion of the right vocal cord was discovered. Clinically, this tumor appeared to be a fibroma.

Under suspension laryngoscopy the lesion, about 3 by 8 mm., was clipped from the vocal cord, and the base was electrocoagulated.

A tissue diagnosis of "neurofibroma" was made.

The preoperative and postoperative mobility of the cords was normal.

Three years later the patient was re-examined. Although there was a slight thickening of the right vocal cord, this did not appear to be a recurrence of the tumor, and no treatment was necessary.

Case 4: A boy, five years old, was presented for examination. His guardian stated that stridor was very noticeable when the boy was asleep.

Examination under suspension laryngoscopy showed a large (2 cm.) soft tumor situated on the posterior surface of the right arytenoid muscle. It displaced the larynx forward and thereby produced partial obstruction of the glottis. The overying mucosa was intact. The lesion was considered too large to permit removal through the mouth.

Accordingly, after preliminary temporary tracheotomy, the lesion was exposed by lateral pharyngotomy and was excised. The postoperative course was uncomplicated.

There was no follow-up examination of this patient, so that the status of the mobility of the vocal cords was not established.

Case 5: A woman, 19 years old, presented herself for examination complaining of "something in the throat" of nine months' duration. She had noted a change of voice, and an inability to sing. She was not really hoarse, but her voice had an unusual quality, as if she was about to clear her throat. She experienced only slight dyspnea on exertion and no dysphagia. A general examination gave negative results.

Examination of the larynx disclosed a smooth, rounded tumor situated in the patient's left aryepiglottic fold, overhanging the glottis and obscuring the left vocal cord. The overlying mucosa was normal in appearance (see Fig. 1).

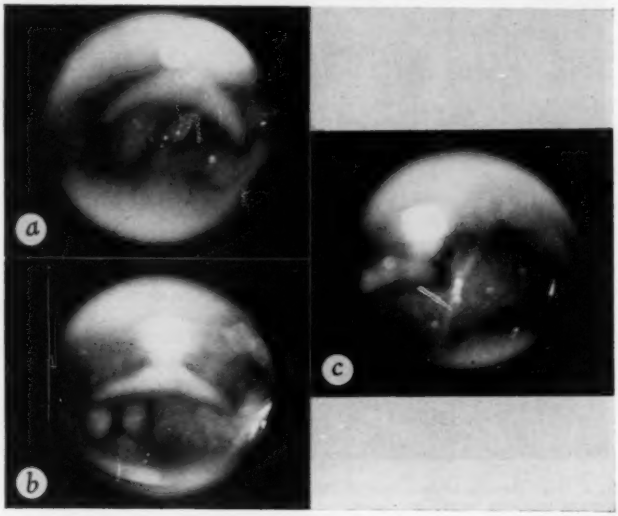

Fig. I. Preoperative mirror view of the larynx of the patient concerned in case 5: A, appearance of the larynx on phonation; B, appearance of the larynx at rest; C, view showing the hypopharynx on the left. Note the rounded tumor mass involving the left aryepiglottic fold, overhanging the glottis to obscure the entire left vocal cord and the posterior portion of the right vocal cord.

The patient was examined under suspension laryngoscopy. The tumor was found to be solid. Because of the size of the lesion, an intratracheal tube was inserted through the mouth to insure an adequate airway. The overlying mucosa was incised down to the tumor. The mucosa was easily reflected from the well-encapsulated tumor, and the tumor was enucleated from its bed. There was practically no bleeding.

The tumor was 2.8 cm. in diameter. Microscopic examination showed it to be a neurofibroma.

The convalescence was uncomplicated. At no time was there evidence of any respiratory obstruction. Examination of the larynx twenty days after the operation disclosed slight edema of the left arytenoid and aryepiglottic regions (see Fig 2). The site of the incision had not entirely healed. There was only slight limitation of motion of the left vocal cord.

Correspondence with the patient's local physician revealed that examination of the larynx ten months postoperatively showed an entirely

normal larynx. Movement of the vocal cords was normal and the line of incision for the removal of the tumor was not evident.

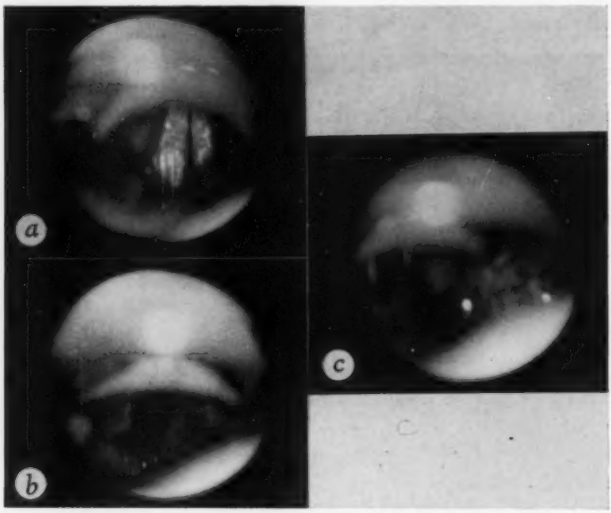

Fig. 2. Postoperative mirror view of the larynx of the patient concerned in case 5: A, appearance of the larynx on phonation; B, appearance of the larynx at rest; C, view showing the hypopharynx on the left. Note the minimal reaction twenty days postoperatively.

SUMMARY.

Neurofibromas involving the larynx may be solitary or a part of generalized neurofibromatosis.

The treatment is surgical removal of the tumor.

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RECOVERY CURVES AND EQUINOXIOUS EXPOSURES IN REVERSIBLE AUDITORY FATIGUE FOLLOWING STIMULATION UP TO 140 DB PLUS.*

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INTRODUCTION.

In spite of scores of articles on the after-effects of acoustic stimulation at intensities just short of those producing permanent damage, quite a number of points still remain obscure. The most extensive study, that of Davis,[1,2] explored the effect of noise and pure tone stimulation on four auditory factors, namely, acuity, loudness, diplacusis, and speech reception. Intensities up to 130 db and durations as long as 64 minutes were used. While this study laid down the broad outlines of stimulation after-effect, it was by the very breadth of the problem forced to content itself with sampling the almost innumerable possible stimulus combinations of frequency, duration, and intensity.

A somewhat more detailed look at the recovery curve following stimulations considerably weaker than those of Davis, *et al*, was provided by Hirsh and Ward.[3] Rather than take the usual clinical audiogram as in the earlier study, these workers continuously recorded the threshold to a particular frequency, disregarding for the moment all other information on recovery. The shape of individual recovery curves was reported for 5-10 representative frequencies after stimulation for three minutes at (1) 500 cps, 120 db sound pressure level (SPL); (2) 500 cps, 100 db; (3) 1000 cps, 100 db; (4) 2000 cps, 100 db; and (5) 4000 cps, 100 db. The usual negative acceleration and wide individual differences were found.

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*From the Naval Medical Research Laboratory

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Unfortunately for us, the intensities and durations used by Hirsh and Ward were not detailed enough to predict (by extrapolation) recovery following some intensities and durations of interest to us. It seemed advisable to study recovery in detailed fashion under a wider range of intensities and durations. The present study summarizes our results thus far.

APPARATUS AND PROCEDURE.

It was desired to be able to stimulate the ear with a fairly pure tone, and within a second or so after the tone was turned off to begin a continuous check on threshold at another frequency. A Hewlett-Packard oscillator was connected to a Permoflux PDR-8 earphone through a MacIntosh amplifier. The same earphone was connected to a Sonotone audiometer. A switch enabled the experimenter to change from one tone source to the other at will.

The desired sound pressure level (SPL) was obtained with the National Bureau of Standards 9A 6 cc. closed coupler, associated microphone, and voltmeter calibrated in dynes/cm.^{2} Repeated settings were obtained with the use of a vacuum tube voltmeter at the earphone terminals.

The function of the audiometer was to obtain pre- and post-stimulation thresholds at the stimulation frequency (750 cps), and at the frequency under examination (1000 cps). Pre-stimulation thresholds were established as the intensity at which (on an ascending series) the subject could hear two consecutive pulses of the audiometer. Pulses were about three-fourth second long, separated by the same duration; intervals were controlled by a photo-electric timer outside the soundproof test room.

After pre-stimulation thresholds were obtained, the desired SPL set, and the fatiguing tone switched to the ear for the desired duration, the experimenter tapped the microphone of a tape recorder and simultaneously threw the switch admitting the pulsing audiometer signal. The audiometer had previously been set to 1000 cps, 20 - 30 db over subject's normal threshold. When, usually after a few seconds, auditory

fatigue had declined to where two consecutive pulses were audible, the subject tapped the microphone, and the experimenter quickly turned the pulse intensity down 5 - 10 db, and waited till the subject again tapped the microphone. By later analyzing the recorded tape, the experimenter could chart the complete course of recovery down to the pre-stimulation threshold. This record was accurate to within a very few seconds. By preliminary exploration, the experimenter sought to select initial audiometer settings and steps of intensity reduction such that the subject's fatigue did not recover faster than the experimenter could turn the dials accurately. For example, if the recovery should proceed to within 20 db of threshold within three seconds, while the dial was still set at 30 db, an error would obviously intrude.

If the subject had not completely recovered after one minute, his threshold was taken once per minute by the same procedure as in the pre-stimulation period, until full recovery.

Three normal-hearing subjects were used, but these were selected from a larger group of the laboratory staff as being among the least susceptible to fatigue. We were not interested for the moment in individual differences nor in the performance of the average subject, but only in changes in the shape of the recovery curve with varying conditions, and in the defining of conditions of stimulation producing equal damage. Our subjects produced relevant data on these questions.

RESULTS AND DISCUSSION.

a. *Auditory area affected by stimulation at 750 cps.* We were interested in recovery especially in the region 1000-2000 cps, a region especially important in speech reception and in several other auditory areas. It was clear from the literature and from some preliminary work of our own, that most damage could be effected in this region if stimulation were at some lower frequency. Fig. 1 gives an entirely typical experiment with a Bekesy-type audiometer on an individual of rather more than average fatigue-susceptibil-

ity. The wide-spread V-shaped frequency effects of a single pure tone are seen, the point of maximum effect remaining as recovery progresses, and the days and probably weeks needed for 100 per cent recovery.

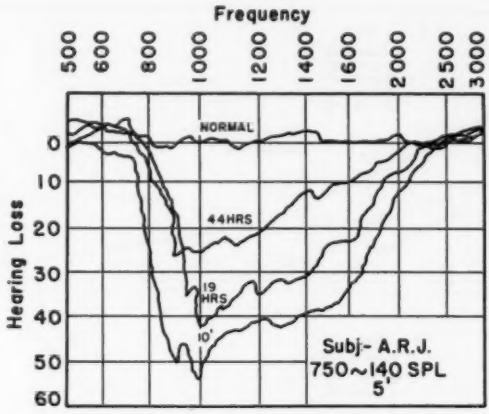

Fig. 1. Stages of Recovery from Auditory Fatigue (Traced from a Bekesy-type Audiogram).

b. *Variability within the individual.* From other preliminary observations it was clear that, for any individual, more than one curve would have to be collected at any given set of stimulus conditions if a true picture were to be presented. It was found that from 3-5 repetitions of the identical stimulus conditions were necessary before stability was reached such that still another repetition would not change the mean curve more than a decibel or thereabouts at any point in time. These repetitions were performed and the resulting mean curve taken as the datum for any subject under the particular conditions.

Two typical examples of variability within the individual are shown in Fig. 2A, B. Since full and complete recovery was allowed between all sessions, and stimulus conditions were identical, we must conclude that there is a considerable inherent variability in an individual's susceptibility to fatigue. The physiology of this variability is quite unknown.

c. *Variability among individuals.* These differences have been emphasized by practically all previous workers and need not be labored here, except to fit the present three subjects into the distribution of a general population. It

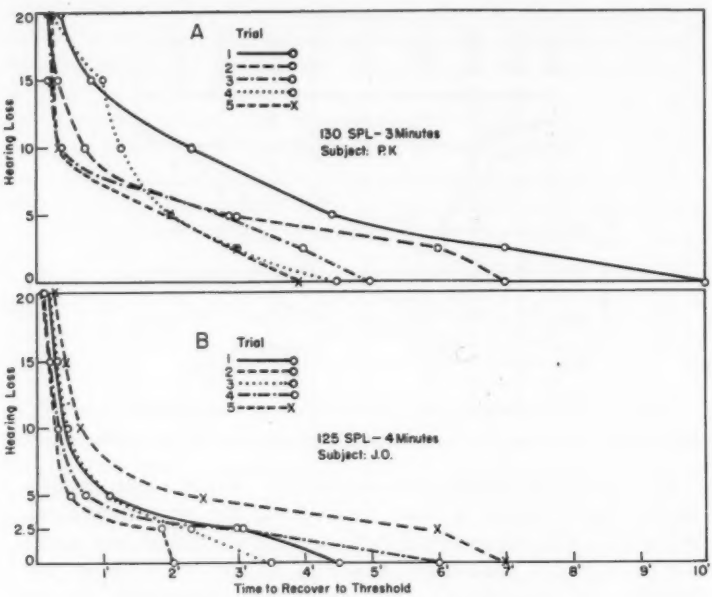

Fig. 2. Variability of Recovery within the Individual.

was stated that of the laboratory staff they were among the least susceptible to fatigue. Fig. 3 compares, for a typical set of stimulus conditions, the recovery curves of these three subjects with a curve from an individual highly suspectible. Two things should be pointed out: (1) the quite wide differences between the two types of subject, the least susceptible recovering in less than seven minutes from stimulation which affects the most susceptible for more than 24 hours; (2) most important for this particular paper, the fact that the three subjects provide quite similar curves, which justify averaging them where desirable.

d. *The shape of the recovery curve.* It was known of course that the recovery curve would take a quite different shape depending on the severity of stimulation, but it was not known what were the precise contributions of duration

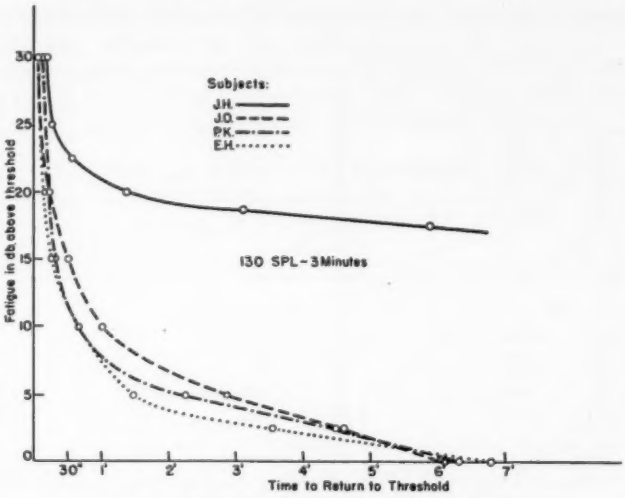

Fig. 3 Individual Variability in Auditory Fatigue.

and intensity separately. Fig. 4A-E shows the average curves for each intensity of stimulation, and at whatever durations were given to all three subjects. (It was not necessary or even desirable, as later data on equinoxious exposures will show, to give each subject exactly the same durations. Nevertheless there was a good deal of overlap, as Fig. 4 demonstrates.)

It is seen that the shape of the curve depends upon both duration and intensity, but is in general a function of some sort of multiplication of duration-times-intensity. Thus, in Fig. 4 one can passably well superimpose the curves which require about the same number of minutes to return to normal (i.e., one index of noxal action), although no two intensities or durations are the same.

The importance of this fact is that one is free to choose quite arbitrarily almost any point on a recovery curve as an index of stimulation effect; if one chooses, for example, the time to return to within 10 or 20 db of normal threshold, or the threshold one, two, or ten minutes after the start of recovery, the whole curve is specified to a first approximation.

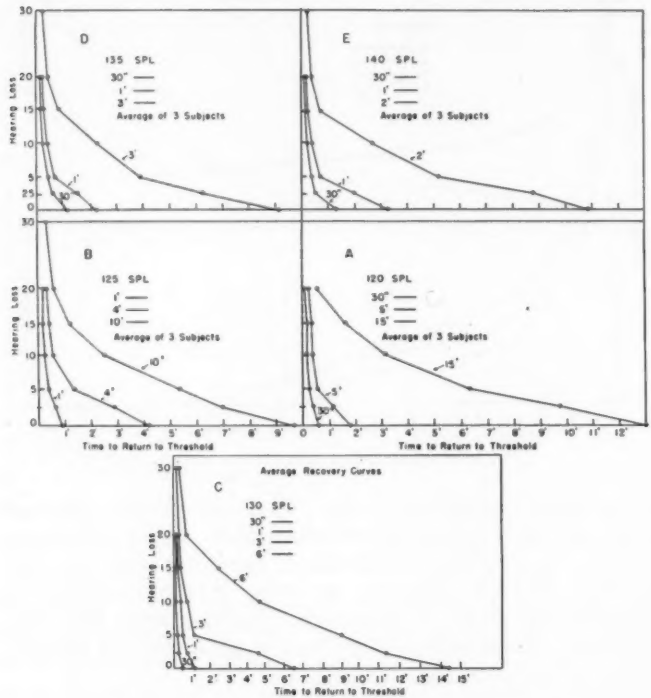

Fig. 4. Typical Recovery Curves at Each Intensity of Stimulation.

A slight difference occurs between the relatively simple shapes of these curves and the recovery curves shown by Hirsh and Ward[3] in which a secondary slight rise in threshold occurred in some subjects after approximately two minutes of recovery. Our experience would indicate that

this occasional rise in threshold is associated with the occasional tinnitus (localized in a fairly specific frequency region) which often follows pure-tone stimulation. All of our three subjects reported intermittent tinnitus frequently, and had we used a continuous tone to seek threshold, as Hirsh and Ward did, our thresholds might have been adversely affected by the intra-aural noise; however, the actual shapes of the recovery curves here presented make it seem likely that our subjects were able to discriminate between any tinnitus and the pulsed signal.

e. *The effect of duration of stimulation.* The effect of duration of stimulation upon the audiogram after two minutes of recovery is shown in Fig. 5. (Similar families

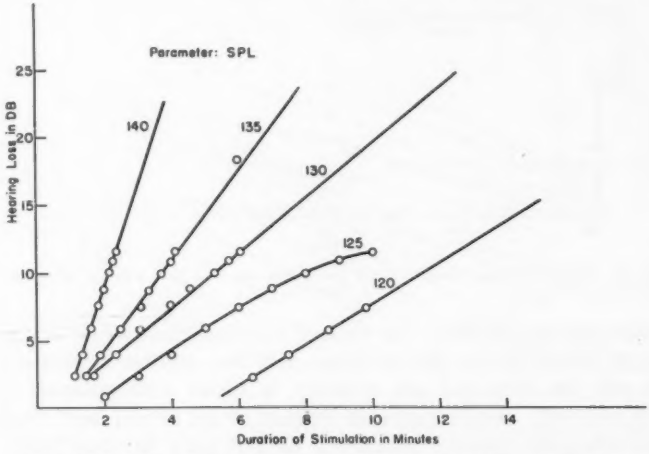

Fig 5. Effect of Duration of Stimulation upon Hearing Loss 2 Minutes after Start of Recovery.

of curves after 20 seconds and one minute of recovery reveal the same state of affairs except of course that fatigue is greater than after two minutes.)

For all intensities used, hearing loss bears a linear relationship to length of stimulation. The regularity makes possible a certain amount of extrapolation to durations

which become wearisome or even dangerous at some intensities. For example, it is probably safe to say that had an intensity of 140 db been applied for six minutes, hearing loss would still be 35-40 db after two minutes of recovery.

f. *The effect of intensity of stimulation.* Similarly, through the range of this study, hearing loss bears a generally linear relationship to stimulus intensity, as shown in Fig. 6. Divergencies from linearity are somewhat more marked than in Fig. 5, but in the main the trend is clear at least

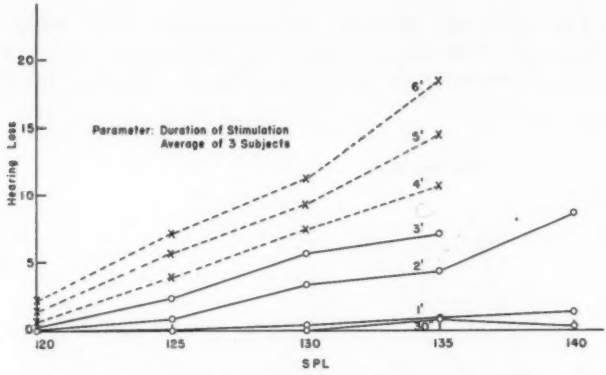

Fig. 6. Effect of Sound Pressure Level upon Hearing Loss Two Minutes after Start of Recovery.

from 120 to 135 SPL. In view of the departure from linearity at 140 SPL for two minutes, and the slighter departures at 135 for five and six minutes, a linear extrapolation to 145-150 SPL would probably introduce an error; but these are intensity regions which we do not care to enter deliberately and have no systematic data on at this time.

g. *Equinoxious exposures.* Of theoretical interest are those intensity-duration combinations which produce equally deleterious effect. Davis, et al[2] are the only ones who have attempted to define these combinations. From their work it is clear that, for equal loss, intensity and time are interchangeable, though in no simple way. Data are given in round numbers, with additional imprecisions in the exact

status of threshold and in the exact time after termination of the stimulus before threshold was taken. It happens that our data are a bit more voluminous and precise on the particular point in question.

Deriving equi-noxious exposures from our data is a simple matter of interpolation. We chose as references the nine average hearing losses after two minutes of recovery following 125 SPL for durations of 2, 3, 4, 5, 6, 7, 8, 9, and 10 minutes. Then, families of curves for individuals similar to Fig. 5 were entered, and the durations at every intensity which produced the reference damages were read. From these data Fig. 7 was constructed. Each line represents a

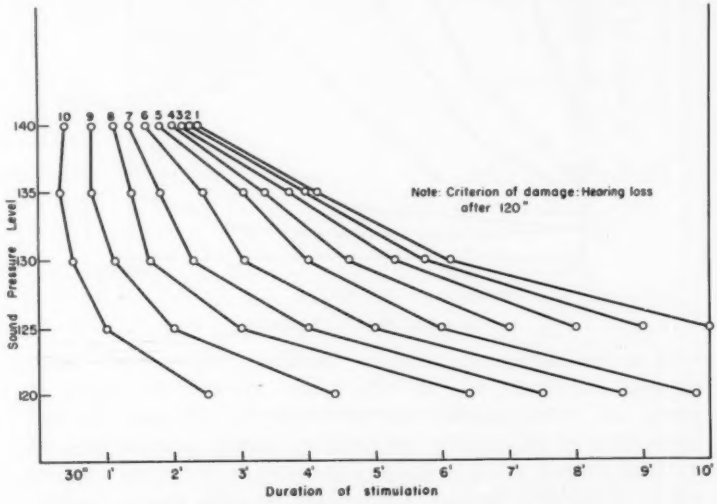

Fig. 7. Equi-Noxious Stimulations (for explanation see Text).

reference damage, and shows which intensity-duration combinations will produce the same loss at the same time stage of the recovery period. Reasonable extrapolations of these curves will begin to delimit the durations at high intensities from which fatigue-resistant subjects should recover in less than a day.

Another way of looking at our data, perhaps a somewhat more practical way than considering only the hearing loss two minutes after start of recovery, is to note time to reach pre-stimulation threshold. Fig. 8 gives equi-noxious curves by this criterion.

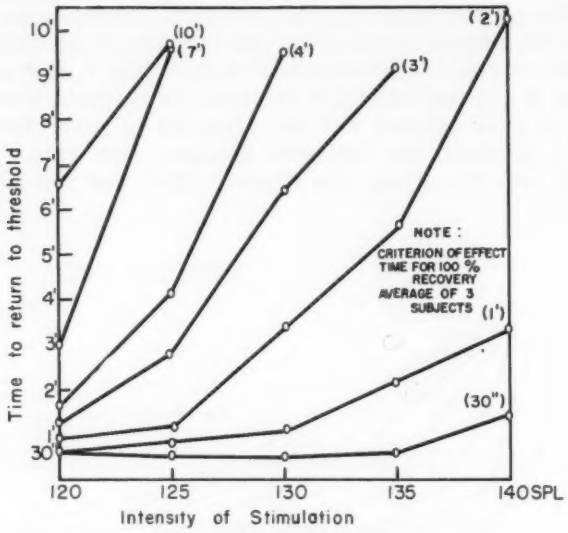

Fig. 8. Equi-Noxious Stimulations.

It is interesting to inquire whether, for various equi-noxious combinations, an equivalent amount of work, in terms of watt-seconds, was delivered to the ear. It was clear that at the higher intensities a much greater amount of total energy must be applied to produce the same noxious effect. Evidently the ear is protected in some special fashion above about 130 SPL so that proportionately longer stimulus durations must be applied. Several opportunities present themselves at higher intensities for the absorption of acoustic energy, but we cannot now differentiate among them.

A closer relation is found between loudness and fatigue than between work and fatigue. Fig. 9 shows the relation between three reference losses, and loudness in millisone-

seconds. These curves show considerable irregularity (partly from inaccuracies in extrapolating loudness at these levels), but in general the trend is for equal millisone-seconds to produce equal noxious action.

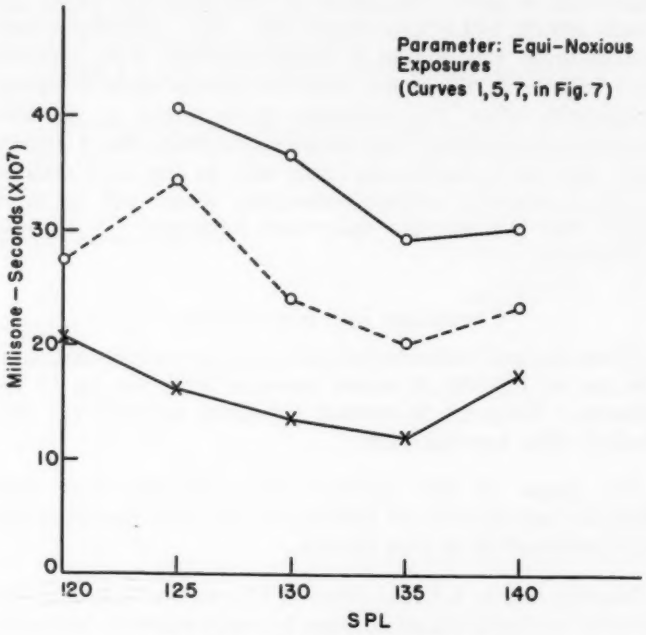

Fig. 9. Relation of Loudness to Hearing Loss.

h. *Practical applications.* Since in daily life human beings are only rarely exposed to pure tones at anything like the intensities used here, the practical importance of these data rest almost wholly upon the demonstration[1] that the effect of a pure tone at, say, 130 SPL, is very close to the effect produced by an octave band of noise of 130 SPL, when the octave is symmetrically placed around the pure tone in question. Thus, the losses produced here at 750 cps may be compared to the losses which would be produced by a band of noise of about 612-1224 cps.

Since one standard set of octave-band filters for analyzing noise contains the band 60-1200 cps, our data can be used by anyone using this filter-set as a first approximation in predicting reversible losses. For example, in a particular industrial situation the noise in the 600-1200 band may reach 120 db SPL. One could with some confidence, now, predict from Fig. 8 that a fatigue-resistant man spending four minutes in this noise would be normal again at speech frequencies after 1½ minutes; if he spent 10 minutes, recovery would occupy 6½ minutes; however, Fig. 3 emphasizes that these conclusions apply only to the most fatigue-resistant subjects, and that the time values will be much longer for the average man and especially for fatigue-susceptibles.

SUMMARY AND CONCLUSIONS.

Three fatigue-resistant subjects were given stimulation of 750 cps at 120-140 db sound pressure level, for up to ten minutes. Recovery to normal threshold at 1000 cps was studied with especial care.

The shape of the recovery curve depends upon both intensity and duration of stimulation, the best prediction being a combination of both factors.

Hearing loss is a linear function of stimulus duration, the function having a different slope for each stimulus intensity. To a less reliable extent, hearing loss is also a linear function of stimulus intensity, when stimulus duration is the parameter.

A family of equi-noxious curves appears from these data, showing the combinations of intensity and duration which produce equally deleterious effects on the auditory threshold.

These results will predict the hearing losses (at frequencies important in speech communication) which are occasioned by being immersed in a noise band of 600-1200 cps for up to 10 minutes at sound pressure levels up to 140 db.

REFERENCES.

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2. DAVIS, H., MORGAN, C. T., HAWKINS, J. E., JR., GALAMBOS, R., and SMITH, F. W.: Temporary Deafness Following Exposure to Loud Tones and Noise. Final Report, Contract OEMcmr-194, OSRD Comm. on Med. Research, 30 Sept., 1943. Available from Office of Technical Services, U. S. Dept. of Commere, No. PBM 57250.

3. HIRSH, I. J., and WARD, W. D.: Recovery of the Auditory Threshold After Strong Acoustic Stimulation. *J. Acoust. Soc. Amer.*, 24:131-141, 1952.

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ACKNOWLEDGMENT.

Much of the material in this study was collected by Patricia Kelsey Perry, whose assistance is greatly appreciated.

THE RESEARCH STUDY CLUB OF LOS ANGELES TWENTY-THIRD ANNUAL MID-WINTER CLINICAL CONVENTION IN OPHTHALMOLOGY AND OTOLARYNGOLOGY, JANUARY 18—29, 1954

For the Ear, Nose, Throat, the guest lecturers will include Dr. Lawrence R. Boies and Dr. Jerome A. Hilger of the University of Minnesota, Dr. Paul H. Holinger of the University of Illinois, and Dr. Lewis F. Morrison of the University of California.

The guest speakers for the Eye program will be Dr. Daniel B. Kirby, Professor of Ophthalmology, New York University, and Dr. Frank D. Costenbader of Washington, D. C.

Other lectures and instruction courses in Otolaryngology and Ophthalmology will be provided.

It is urgently advised that you write for reservations at an early date to Mr. H. M. Nickerson, Manager of the Elks Club, Parkview at Sixth Street, Los Angeles 5. He will endeavor to arrange suitable quarters for you in a conveniently located hotel, and will confirm the reservation by return mail.

The first week will be devoted to the Ear, Nose, Throat—from Monday to Saturday, January 18—23. The Eye week will be from Monday to Friday, January 25—29. Those who confine their work to only one of the specialties may complete either subject in one week.

An evening of entertainment is planned for members attending the Convention, their wives and friends. The women's committee has arranged for a luncheon for the wives of attending members, and a Dinner the evening of the Society Banquet. There will also be a cocktail party and organ recital honoring our Guest-Speakers and their wives. The dates for these events will be announced later.

Each applicant must be a member, in good standing, of the American Medical Association in order to become eligible for

attendance at the course. Send your fee of $100.00 to Pierre Violé, M. D., Treasurer, 1930 Wilshire Boulevard, Los Angeles 5. If anything prevents your attendance, this fee will be returned to you. The fee for the Clinical Course will include the cost of all luncheons and the cocktail party. Make checks payable to Mid-Winter Clinical Convention.

WANTED — HEAD OF E. N. T. DEPARTMENT AT CHRISTIAN MEDICAL COLLEGE, VELLORE, SO. INDIA

The Vellore Christian Medical College, So. India—eighty miles west of Madras City—is an international, union enterprise of the mission boards of Protestant Church denominations in Canada, Britain, Australia, and the United States. The medical school, which was originally established in 1916, is affiliated with the University of Madras, which confers the M.B.B.S. degree—Bachelor of Medicine, Bachelor of Surgery, equivalent to M.D. in U. S. A. The medical college hospital has 550 beds, a dozen of which are allotted to the department of otolaryngology. The ENT out-patient department has a daily attendance of 50 to 60 patients.

There are 175 students enrolled in the five medical classes. The program of teaching comprises 12 lectures to the fifth-year students, sections for clinical work, rounds and clinico-pathological conferences. The total faculty consists of about sixty full-time teachers, of whom about a third are Westerners. On the ENT staff there are an American professor—now giving part time and desiring to withdraw—an associate who has the diploma in Oto-rhino-laryngology of the University of Madras, an intern and two residents.

There is a large volume of clinical and operative work, dealing with a great variety of conditions—some peculiar to Southern Asia, many the result of neglect and the lack of sulfonamides and antibiotics in the villages.

The applicant should be a member of a Protestant church, a

Diplomate of the American Board of Otolaryngology, have three years of teaching experience—training of interns and residents in a university-affiliated hospital accepted—and willing to serve for a period of at least three years, on a very modest salary.

Further details may be obtained from Miss Sarah Scudder Lyon, Secretary, Vellore Christian Medical College Board, 156 Fifth Avenue, New York 10, N. Y.

A RIDDLE:

WHAT CAN YOU GIVE AWAY, BUT STILL HAVE?

The answer is *blood!* You can donate a pint to the National Blood Program but still have all your blood . . . because your body makes more to take its place. It's just that simple.

As an average healthy person, you have 12 to 13 pints of blood. Your blood is constantly being replenished. The removal of a pint calls for no special effort on the body's part. The liquid part is replaced almost immediately, the cells and minerals after a few days. Donating a pint of blood is comparable to an afternoon's exercise. A person in good health can do either safely.

So, you can give . . . again and again . . . actually to gain back every drop. When you donate a pint of blood, the only feeling you have is one of satisfaction in helping someone live. A few minutes of your time may mean a lifetime to someone else.

To meet the nation's need for whole blood and derivatives, the National Blood Program must receive the voluntary donation of over 5,000,000 pints this year. To Federal Civil Defense for building reserves of serum albumin, to accident victims in need of whole blood transfusions, to wounded serv-

ice men in need of shock units of serum albumin, and to children exposed to polio in need of the blood derivative gamma globulin, *your blood is vital.*

Your donation means so much and costs you absolutely nothing. Your blood spells life and health to so many without your really losing a thing. Call your local Red Cross, Community or Armed Forces Blood Donor Center today and schedule *your* donation of blood.

AMERICAN OTOLOGICAL SOCIETY, INC. AWARDS ITS 1953 GOLD MEDAL.

The American Otological Society, Inc., awarded its gold medal and a scroll to Dr. Julius Lempert at its annual meeting in New Orleans on May 1, 1953.

The award was given to Dr. Lempert for his outstanding contribution to the surgery of the temporal bone. The scroll which accompanied the gold medal bore the following inscription:

"The American Otological Society, Inc.

1953

Award of Merit Gold Medal

Presented to Julius Lempert, M.D.

"Your investigations, carried on through trials which would have discouraged a lesser figure, have so perfected the fenestration operation that your technique is accepted throughout the world and is regarded as a boon to mankind.

"Your remarkable studies, superior surgery and inspiring teaching mark you as the one who has made the greatest single contribution to otology in this generation."

LIème CONGRES d' OTO-RHINO-LARYNGOLOGIE

The LIème Congrès Francais d'Oto-Rhino-Laryngologie will be held in the Grand Amphithèâtre of the Faculté of Médecine of Paris, October 26, 29, 1953, Mr. le Docteur Louis Leroux, of Paris, is president, and Mr. le Professeur Léon Binet, Director of the Faculté of Médecine, Paris, is honorary president.

During the Congrès, operations will be observed at the various hospitals. The program will begin on the morning of October 26. There will be an exhibit of instruments in the grand hall of the Faculté of Médecine.

For further details address Dr. H. Flurin, secrétaire général - Cauterets (Hautes Pyrénées), or Dr. H. Guillon, secrétaire général adjoint - 6, avenue Mac Mahon, (XVIIe).

AMERICAN HEARING SOCIETY

Plans to move national headquarters of the American Hearing Society from the nation's capital to Columbus, Ohio, are announced by Thomas L. Tolan, M.D., Milwaukee otologist, president of the agency. No definite date has been set for this change but the Society wants the move completed as soon as possible.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

July 1, 1953.

Audicon Models 400, 415, 530 and 615.
Manufacturer: National Earphone Co., Inc., 20-22 Shipman St., Newark 2, N. J.

Auditone Model 11.
Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Symphonette; Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 149 Church St., New York 7, N. Y.

Fortiphone Models 19-LR; 20A; 21-C and 22-C.
Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.

Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico UE-Atomeer; Maico Quiet Ear Models G and H; Maico Model J; Maico Top Secret Model L.

Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.

Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Classic Model T9; Microtone Model T10; Microtone Model T612; Microtone Model 45.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Cub Model C; National Cub Model D (Duplex); National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Normatone Model C.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-2; Otarion Model G-3; Otarion Model H-1.

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40. Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Mite); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp.

Silvertone Model J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.

Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal; Model Cameo.

Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).
Manufacturer: A. M. Brooks Co., 64 S. Bonnie Brae St., Los Angeles 5, Calif.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

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President: Dr. Frederick T. Hill, Professional Bldg., Waterville, Me.
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Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Statler Hotel, Boston, Mass., May 16-17, 1954.

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Meeting: Statler Hotel, Boston, Mass., May 25-26 (Afternoons only), 1954.

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Meeting: Palmer House, Chicago, Ill., Oct. 11-17, 1953.

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Secretary: Dr. Lawrence R. Boies, Med. Arts Bldg., Minneapolis 2, Minn.
Meeting: Palmer House, Chicago, Ill., Oct. 11-17, 1953.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

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Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Palmer House, Chicago, Ill., Oct. 16, 1953.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Justo M. Alonso, Montevideo.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fourth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. Ricardo Tapia Acuna, Mexico City.
Time and Place: Feb. 28 to Mar. 4, 1954, Mexico City.

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Secretary of Section on Otolaryngology: Max E. Pohlman, M.D.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:00 P.M., fourth Monday of each month from September to June, inclusive—Otolaryngology Section; 6:00 P.M., first Thursday of each month from September to June, inclusive—Ophthalmology Section.

THIRD LATIN AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

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Secretary General: Dr. Victorino Marquez Reveron.
Secretary of Assemblies: Dr. Cesar Rodriquez.
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AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

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Secretary: Dr. Louis Joel Fleit, 66 Park Ave., New York (16), N. Y.

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Secretary and Treasurer: Dr. Geo. B. Ferguson, Durham, N. Car.
Meeting: Charleston, S. Car., Sept. 13-16, 1953.

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Vice-President: Dr. David S. Asbill, Columbia, S. Car.
Secretary-Treasurer: Dr. Roderick Macdonald, Rock Hill, S. Car.
Meeting, Joint: Charleston, S. Car., Sept. 13-16, 1953.

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Meeting: Honolulu, 1954.

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President: Dr. D. E. S. Wishart, 170 St. George St., Toronto, Ontario.
Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place:
Time:

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Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Andre Soulas, Paris, France.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa. U. S. A.
Meeting: 3rd International Congress of Broncho-Esophagology.
Time and Place: September or October, 1954, Lisbon, Portugal.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER — CAMPINAS.

President: Dr. Joao Penido Burnier.
First Secretary: Dr. Gabriel Porto.
Second Secretary: Dr. Roberto Barbosa.
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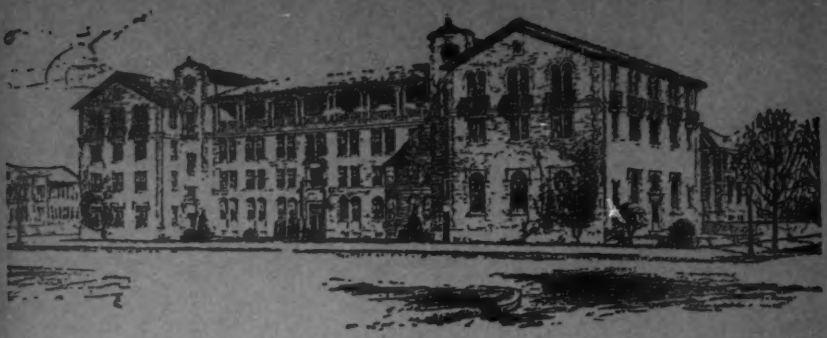

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